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WHAT THE EAR DOES NOT HEAR THE BRAIN CANNOT INTERPRET

The ear is no more than the receiver of sounds; it is the brain which interprets them.[1] If the delicate apparatus which collects sound waves is defective, no impulse may reach the brain.

To a lesser degree the effect is the same if hearing loss is partial. The brain strives to interpret isolated sounds, succeeds only imperfectly. It is like trying to piece together a jigsaw puzzle in which some pieces are missing, like trying to understand a strange name over the telephone. Only when endeavoring to understand unfamiliar words or phrases is the hearing person aware that telephone transmission is only 70 per cent effective (a satisfactory transmission according to engineers [2]).

The hard-of-hearing, however, are constantly confronted with the problem of interpreting partially heard words or phrases. When one considers that patients with high tone deafness may be unable to distinguish between the ring of a bell and the tap of a drum,[3] some slight idea may be obtained of the magnitude of the problem facing the hard-of-hearing. No wonder it sometimes seems easier to retreat into a world of silence where no effort to hear is needed.

This tendency must be recognized and combatted by all the physician's knowledge and art, for withdrawal from life can have nothing but bad effects. Human beings need social intercourse with their fellows to remain emotionally normal.

SPEECH DEPENDS ON HEARING

Speech is a "march of events, acquired step by step in the life of the individual." [4] Persons with normal hearing take these steps so gradually they hardly realize that speech is really the interpretation of sounds and their imitation. Sound alone is meaningless. The Wild Boys of India, living with jungle animals without human contacts, were normal physically and mentally, yet when they were brought back into civilization they never were able to achieve a real grasp of human speech.[5] Their hearing centers had remained dormant.

So, to a lesser extent with any child. If his hearing is defective it is evident that his speech must be adversely affected.[2] How, indeed, can he be expected to reproduce correctly words he has never distinctly heard? His hearing center, too, may remain dormant, and precise language may always remain beyond his grasp.

The physician who sees the child for any of the numerous illnesses of the young, may, if he is alert to the possibility of hearing loss, be the first to recognize it. Such early recognition will be of inestimable value in directing the child's life.

The hard-of-hearing pre-school child must be the focus of an educational program. It is especially under three years that the educational treatment should start.[6] Physicians must strive to overcome the current apathy toward the hard-of-hearing child.[6] Theirs is the responsibility for providing the means by which residual hearing may be utilized, so the child may develop normal speech patterns.

LOSS OF CURRENCY OF WORDS

Adult speech, too, suffers when hearing losses develop.[6,7,8] Those who have normal hearing are not aware that *spoken* language, even more than *written* language, never stops growing and changing, that old words attain new connotations and even new meanings, as well as new associations.[7] To the hard-of-hearing words are far more static—they often mean to them only what they did before the hearing became diminished. Only by the most constant and exhausting attention can these people keep abreast of current words. Seeing a new word in print is not enough. What does penicillin sound like when it is spoken? When this word first appeared in our common language many pronounced it pen-iś-ilin. And how would you pronounce "Rh factor" if you had only seen the term and not heard the words?

The incorrect pronunciations of the youngster whose reading vocabulary exceeds his speaking vocabulary is well recognized. Those whose hearing is appreciably below normal have an even more difficult problem; they neither know how to pronounce a new word, nor can they easily recognize it when it is spoken. Moreover, the adult is far more sensitive than a child to receiving correction of his mispronunciations and in asking the meaning of unfamiliar sounding words.

The longer the hearing defect remains uncorrected the more difficult it becomes for the hard-of-hearing to take part in a lively conversation rich in new words; the less likely he is to pick up, to understand, unfamiliar words; the more diffident he becomes about pronouncing words he has not heard. So more bricks are added to the barrier between him and his friends.

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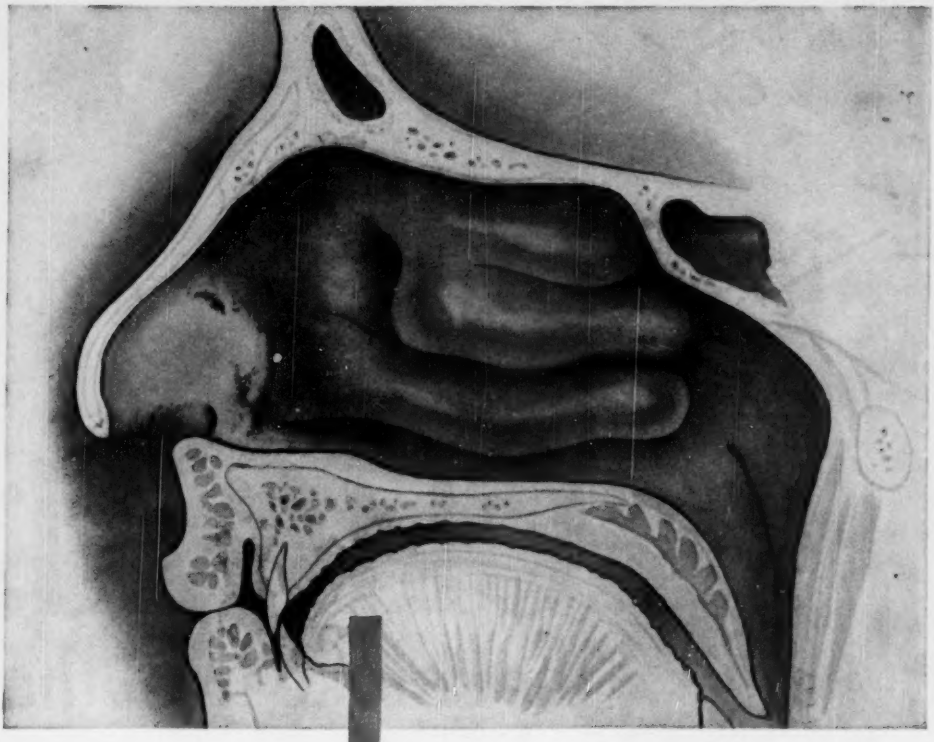

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*Anderson, J. and Steele, C.: *Use of Nitrofuran Therapy in External Otitis*, Laryngoscope 58:1279, 1948 • Douglass C.: *The Use of Furacin in the Treatment of Aural Infections*, Laryngoscope 58:1274, 1948 • Reardon, H.: *Unpublished results.*

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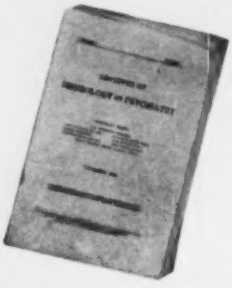

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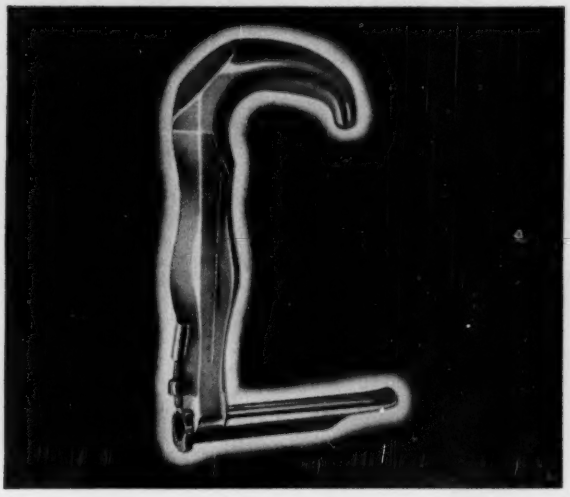

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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VOLUME 51 APRIL 1950 NUMBER 4

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COLLAPSED ALA

Pathologic Physiology and Management

SAMUEL FOMON, M.D.

JOSEPH G. GILBERT, M.D.

NEW YORK

ARMAND L. CARON, M.D.

WORCESTER, MASS.

AND

SAMUEL SEGAL Jr., M.D.

SPRINGFIELD, MASS.

THE PURPOSE of this paper is to discuss a type of inspiratory nasal obstruction in which the lateral cartilaginous wall is sucked against the septum, partially or completely shutting off the onward passage of air. The condition is commonly referred to as "collapsed" or "collapsing" ala. Perhaps the term "cartilaginous inspiratory obstruction" would better represent the syndrome.

The clinical picture is characteristic (fig. 1). If the collapse involves both upper and lower lateral cartilages, or only the latter, the entire cartilaginous walls collapse and the nostrils appear slitlike with each inspiration. If the collapse is limited to the upper lateral cartilages, the central portion of the nose assumes a pinched appearance, the nostrils dilate markedly and the exaggerated activity of the alar muscles is clearly visible. In either case the degree of collapse is in direct proportion to the depth of the inspiratory effort. Occasionally, the closure is so abrupt as to be evidenced by a distinct audible click as the cartilaginous wall "slaps" against the septum. On expiration, the alae assume their normal positions and the current passes out without hindrance. Frequently patients volunteer the information that relief is afforded by drawing the cheek adjacent to the ala outward and upward or by raising the tip of the nose.

The relation between the degree of collapse and the depth of inspiration is of considerable diagnostic importance. Generally speaking, if the collapse occurs during quiet breathing, the fault is probably an inherent weakness of the cartilage, which is so yielding as to be unable to resist the normal negative pressure on inspiration. If, on the other hand, collapse occurs only on deep inspiration, the cartilages need not necessarily be at fault. Here they may collapse secondarily to con-

strictions in the vestibule, in which the narrowed passage, by impeding the entrance of air, increases the negative pressure above the constriction to such a degree that the cartilage, even though normal, may be unable to resist it (fig. 9). Increasing the patency of the aperture—for example, compressing a thickened columella between the blades of a forceps or forcing a concave ala outward with a probe—will frequently relieve the collapse temporarily by normalizing the intranasal pressures.

Inspection and palpation of the vestibule will reveal the faulty architecture and/or the abnormal consistency of the cartilage responsible for the collapse. To obtain a clear view, the vibrissae are clipped

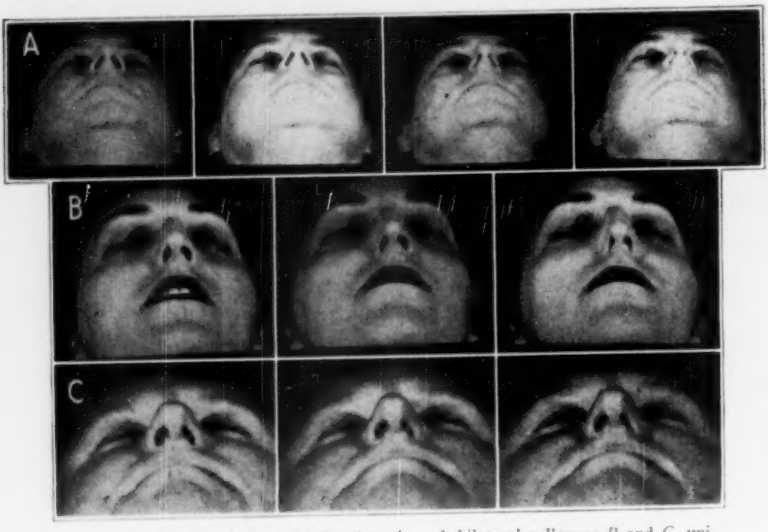

Fig. 1.—Motion picture of collapsing ala. *A*, bilateral collapse; *B* and *C*, unilateral collapse.

and the parts cleaned. In no circumstances should a speculum be used in this examination, for it distorts and conceals the anatomy.

ANATOMIC FACTS

A cursory review of a few anatomic facts pertaining to the structures involved in collapsing ala is essential to a clear understanding of the management. The cartilaginous vault is the forward continuation of the bony piriform opening and is composed of the septal cartilage with its two winglike projections, the upper lateral cartilages and two independent horseshoe-shaped structures, the lower lateral cartilages.

The upper lateral cartilages (fig. 2) spread outward and downward from the ventral (anterior) border of the septum in the shape of inwardly arched triangular plates. They form approximately the middle third of the nose and are important elements in the maintenance

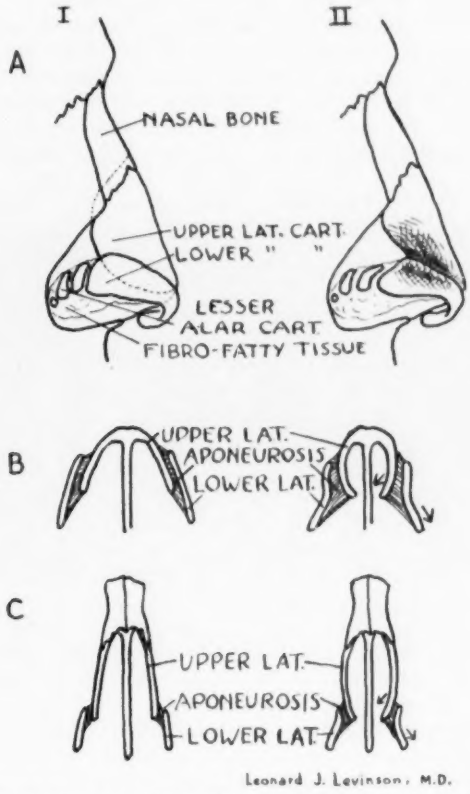

Fig. 2.—Nasal cartilages during quiet and deep respiration. I, quiet inspiration. *A*, lateral view; *B*, horizonal section, and *C*, coronal section. II, deep inspiration. *A*, lateral view; *B*, horizontal section and *C*, coronal section, showing simultaneous inward swinging of upper lateral cartilages and outward swinging of lower lateral cartilages. The intervening aponeuroses prevent abutment of upper lateral cartilages against septum.

of the nasal contour in this location. The ventral (anterior) borders are thick and directly continuous with the septal cartilage. The cephalic (superior) borders are attached to the under surfaces of the nasal bones

and ascending processes of the maxillas. The caudal (inferior) borders extend 2 to 3 mm. beneath the upper edges of the lower lateral cartilages and are joined to them by fibrous aponeuroses (fig. 2). The lower lateral cartilages, each consisting of a mesial and a lateral crus, encircle the nostrils and assist in maintaining their patency. The mesial crura are united to the caudal edge of the septal cartilage by an interposed membranous septum. The lateral crura overlap the upper lateral cartilages. Dorsally (posteriorly) they extend toward, but fall short of, the piriform opening, becoming lost in a dense sheet of fibrous tissue. The accessory alar cartilages, considered by von Mihalkovics[1] as clipped-off portions of the lateral crura, are small, vary in number and are found embedded in fibrous tissue in the wings of the nose (fig. 2).

RESPIRATORY MECHANISM

The importance of the nasal cartilages in respiration can best be appreciated by a brief review of the respiratory mechanism. The lung is in free communication with the outside air. Consequently, at the end of inspiration or expiration the pressure within the lung is equal to that of the atmosphere. On inspiration the respiratory muscles contract and cause the thoracic cage to increase in size. Temporarily, the pressure of the rarefied air in the enlarged space becomes less than that of the atmosphere, and air rushes into the lung. The extent to which the pressure falls will depend, naturally, on the amplitude of the muscular contractions and on the sizes of the opening to the exterior. On expiration the subsequent relaxation of the muscles brings about a diminution in the size of the thoracic cage. Now the pressure of the air compressed within the smaller space becomes temporarily greater than that of the atmosphere, and air rushes out. In this process the lung is a passive agent, merely following the movements of the thoracic box. External respiration, then, is an expression of muscular activity. This activity is exemplified in anterior poliomyelitis, in which paralysis of the respiratory muscles causes death from asphyxia. The "iron lung" is not a lung at all. Actually it is an "iron muscle."

The placing of a load on a muscle sets up, in a manner not yet understood, some local stimulus which causes the muscle to accomplish more work. Up to a certain point, the greater the load, the greater the strength of the muscular contraction.[2] Drury[3] referred to this reac-

1. von Mihalkovics, V.: Anatomy and Embryology of the Nose and Its Sinuses, in Heymann, P.: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hölder, 1900, vol. 3, pt. 1.

2. Fenn, W. O.: Mechanics of Contraction, in Bethe, A.; Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie mit Berücksichtigung der experimentellen Pharmakologie, Berlin, Julius Springer, vol. 8, pt. 1, 1928. Fenn, W. O.: Isotonic Contractions in Muscle, Symp. Quant. Biol. **4**:233, 1936.

3. Drury, D.: Personal communication to Dr. Victor Goodhill.

tion as "the law of initial tension." For the muscles of respiration, the load is represented by the resistance offered to the current by the respiratory canal. Obviously, more work on the part of the respiratory muscles is required if a given volume of air per unit of time is taken in through the long, narrow, sinuous canal of the nose than through the shorter, more spacious and more direct oral route. It is interesting to note that, despite the shortness of the nasal canal as compared with the rest of the respiratory tract, it offers, according to Rohrer,[4] nearly 50 per cent of the total tubular resistance (i.e., roughness of wall, speed and specific resistant constants). The resistance thus offered by the nose to the incoming air constitutes a major part of the optimal load. Perhaps in this lies one of the chief benefits to be derived from nasal breathing.

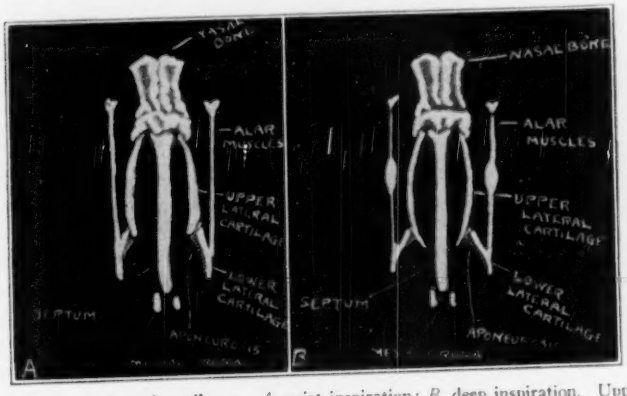

Fig. 3.—Action of cartilages. *A*, quiet inspiration; *B*, deep inspiration. Upper lateral cartilages approach septum, narrowing aperture. Contraction of muscles causes lower lateral cartilages to swing outward. Tension on aponeuroses prevents cartilages from abutting septum.

So important is the optimal load to proper breathing that even mouth breathers unconsciously try to emulate the physiologic nasal resistance to the air current by narrowing the lips and teeth to a small chink and applying the tongue to the palate during respiration. Likewise, patients suffering from a deviated septum without complete obstruction usually breathe better on the narrow side, where the resistance is greater. On the same basis, the hypertrophy of the conchae on the wide side may represent a compensatory attempt to increase the resistance. On the other hand, after a too generous submucous resec-

4. Rohrer, R.: Current Resistance in the Human Respiratory Passages and the Effect of the Irregular Ramification of the Bronchial System on the Course of the Air in the Various Pulmonary Regions, Arch. f. d. ges. Physiol. **162**:225, 1915.

tion, the resistance to the current may have been so completely removed as to nullify the purpose of the operation.

As previously mentioned, pressure fluctuations within the lung depend not only on the amplitude of the inspiratory movements but also on the size of the opening to the exterior. The narrowest portion of the respiratory tract lies between the caudal (inferior) end of the upper lateral cartilage and the septum (ostium internum). Alterations in the size of this aperture will, in large measure, determine the degree of pressure behind it. With the body at rest, the narrowness of the nasal canal offers the optimal load for the respiratory muscles, and thus no extra accommodation is needed. During exercise, however, the respiratory muscles must contract more forcibly to supply the increased need for oxygen, and this calls for an increased load on the muscles. Furthermore, this load must be sufficiently flexible to cope with the varying oxygen needs. The upper lateral cartilages serve this double purpose to a large degree. On inspiration they swing toward the septum, narrowing the aperture and, thereby, increasing the load on the muscles. The muscles, working against resistance, must contract more forcibly, thus providing for a deeper inspiration (fig. 3). In other words, the upper lateral cartilages serve as resistance regulators in the air current—a kind of living rheostat. Uddström[5] came to the conclusion that in a normally shaped nose the upper lateral cartilages were responsible for 70 per cent and the conchae for 30 per cent of the total nasal resistance. He found that in an abnormally wide nose the resistance was supplied, in the main, by the action of the upper lateral cartilages; whereas in a narrow nose the resistance was furnished by the cavernous tissue of the conchae.

On deep inspiration the aperture between the upper lateral cartilage and the septum narrows down to a very small opening, but complete closure is prevented by the checkrein action of the lower lateral cartilage in the following manner: As air is drawn into the nose, the dorsocaudal (posteroinferior) portions of the alae flare outward and upward, putting the intervening aponeuroses on stretch and limiting the inward movement of the upper lateral cartilages. This position is maintained by the lesser alar cartilages, which slip under the upper lateral cartilages like doorstops and take the strain off the muscles (fig. 2).

The muscles responsible for this action (fig. 4) are (1) the dilator naris anterior, which contracts and brings about a closer relation between the upper and lower lateral cartilages; and (2) the caput angu-

5. Uddström, M.: Nasal Respiration: A Critical Survey of Some Current Physiological and Clinical Aspects on the Respiratory Mechanism with a Description of a New Method of Diagnosis, Acta oto-laryng., 1940, supp. 42, p. 3; The Importance of the Conchae for the Resistance Within the Normal Nose, ibid. **28**:364, 1940.

lare and dilator naris posterior, which take hold of the dorsal and ventral ends of the alae respectively and draw them away from the septum. The importance of these small muscles is illustrated by van Dishoeck [6]. In the case of a patient with a unilateral facial paralysis, he compared the nasal resistance of the normal and the paralyzed sides by recording the pharyngeal pressures. In quiet inspiration, with a respiratory depth

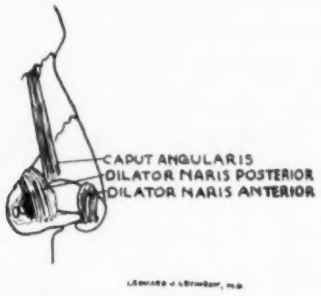

Fig. 4.—Muscles responsible for alar movement.

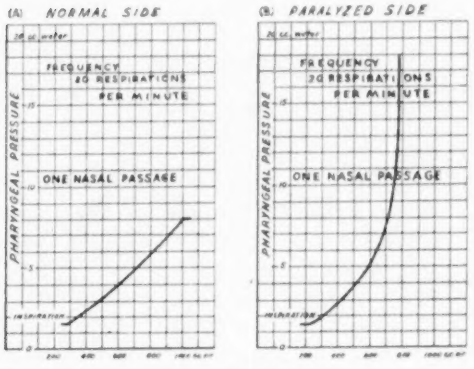

Fig. 5.—Nasal resistance to current recorded by pharyngeal pressure. *A*, normal side; *B*, paralyzed side, showing effect of collapse. See text.

of 400 cc., the negative pressure in the pharynx on both the paralyzed and the nonparalyzed side was about 2.5 cm. of water. But on forced inspiration, with a respiratory depth of 800 cc., the resistance on the normal side was found to be only minus 5.5 cm. of water; whereas on

6. van Dishoeck, H. A. E.: Electrogram of the Alar Muscles and Nasal Resistance Curve, Acta oto-laryng. **25**:285, 1937.

the side devoid of muscular action it was minus 19 cm., showing the effect of collapsed ala on deep inspiration (fig. 5).

FUNCTIONS OF THE REGULATORY MECHANISM OF THE NASAL CARTILAGES

Better Ventilation of the Lung Through Increase in Depth of the Respiratory Effort.—For example, in mouth breathing, in which less effort is required on the part of the respiratory muscles to bring air into the lung, the respirations are shallow, with a consequent deficiency in

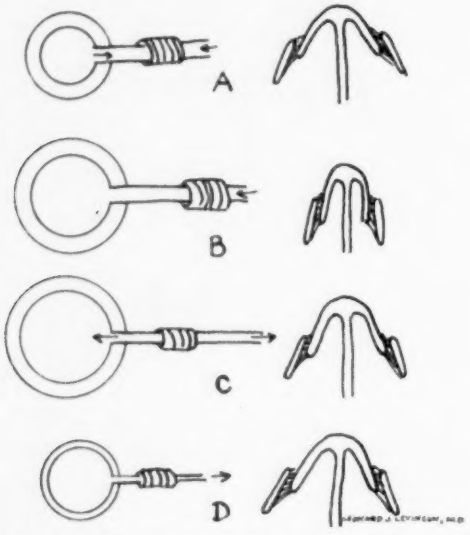

Fig. 6.—Effect of nasal cartilages on air mixing in alveoli. *A*, beginning of inspiration; *B*, end of inspiration; *C*, beginning of expiration, and *D*, end of expiration. See text.

lung ventilation. As compensation for this deficiency, the respiratory rate increases. The increased rate, however, cannot make up for a deficiency in depth, since the capacity of the dead space (i.e., the air in the tracheobronchial tree, averaging 150 cc. and subserving no gaseous exchange function) remains the same irrespective of the depth of respiration. For example, with a normal inspiration of tidal air (500 cc.), 150 cc. occupies the dead space and 350 cc. enters the alveoli, where the gaseous exchange takes place. Thus, at the rate of 15 respirations per minute, the total intake would be 7,500 cc., and the air available for

respiration would be 350 by 15, or 5,250 cc. In a shallow respiration, with an intake of 200 cc. at the rate of 50 per minute, the total intake would be 10,000 cc.—an amount considerably greater than normal. Yet the air available for oxygenation would be only 50 by 50, or 2,500 cc.—an amount less than half the normal.[7]

Better Utilization of Inspired Air.—As mentioned previously, the depth of a respiration is in direct proportion to the amplitude of the muscular contraction. But, since the gaseous exchange between the alveolar air and the blood is a mechanical process of diffusion and requires a certain amount of time for equilibration, the depth is not necessarily in direct relation to the aeration value. Obviously, an excess of air in the alveoli due to an unduly rapid renewal could be utilized only in part, and the balance would be wasted. While the waste of air in itself is of little importance, the excessive work on the part of the muscles required for this rapid renewal is important, for it represents an extreme waste of energy, when one considers that the respiratory movements even in normal breathing are repeated about 1,000 times an hour. The nasal cartilages obviate this waste of air and energy by proportioning the amount of air that enters and leaves the lung in a given unit of time with the muscular effort.[8]

Assistance in the Process of Mixing.—At the start of inspiration, the circular muscle fibers around the bronchioles relax and allow the bronchioles to dilate, with a consequent fall in pressure. Their filling is retarded, however, by a narrowing of the nasal passage caused by the approximation of the upper lateral cartilages to the septum. At this time the pressure in the alveoli is higher than in the dilated bronchioles, since up to now the pressure in the pleural cavity has not been reduced to any evident degree. Therefore, air will rush out of the alveoli into the bronchioles, where it will mix with the supply coming in from the atmosphere. As inspiration proceeds, the thorax enlarges and the diaphragm descends. The resultant negative pressure causes the mixed air in the bronchioles to be forced into the distended alveoli (fig. 6).

At the beginning of expiration the muscle fibers of the bronchioles contract. The space between the upper lateral cartilages and the septum, being the narrowest part of the respiratory tract, offers obstruction to the exit of air, and, since the pressure on the outer walls of the alveoli is still low (because relaxation of the thoracic and abdominal walls has only just commenced), some of the air from the bronchioles is forced back into the air sacs. At the end of expiration the alveoli diminish in

7. Carpenter, T. M., and Lee, R. C.: Influence of Glucose and of Fructose on Effective Dead Space in Human Respiration, Am. J. Physiol. **104**:10, 1933.

8. Mink, P. J.: Physiologie der oberen Luftwege, Leipzig, F. C. W. Vogel, 1920.

volume and the air passes out into the bronchioles. At the beginning of the next inspiration the bronchioles relax and the cycle is repeated.

To summarize, the air flows from the alveoli to the bronchioles at the beginning of inspiration; from the bronchioles to the alveoli during the remainder of inspiration; from the bronchioles to the alveoli at the beginning of expiration, and from the alveoli to the bronchioles during the remainder of expiration.

In oral breathing the filling and emptying of the bronchioles is so rapid, owing to the large caliber of the canal, that the pressure is too quickly equalized to allow adequate mixing of air.[9]

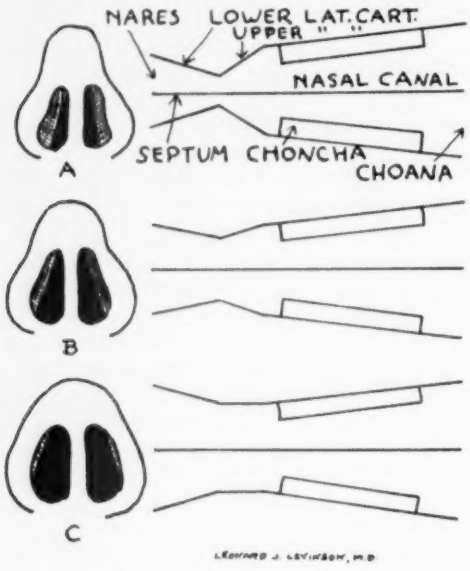

Fig. 7.—Action of cartilages in regulation of intranasal pressure (modified from Proetz[10]). *A*, constricted ostium internum; *B*, normal relation of ostium internum and choana, and *C*, abnormally wide ostium internum.

Assistance in Regulation of the Rhythm of Respiration.—As has been stated, the resistance offered to the respiratory current in nasal breathing will cause air to enter and leave the lung more slowly and evenly than in oral breathing. Accordingly, the impulse calling for inspiration and expiration (Hering-Breuer) will be delayed, and the inspiratory phase will pass over without sharp differentiation and almost

9. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929.

unnoticeably into the expiratory phase. When the nasal cartilages are eliminated from participating in respiration, as in oral breathing, the rhythm is more rapid, at times jerky, and the respiratory pause is longer.

Regulation of Intranasal Pressures.—The size of the space between the upper lateral cartilage and septum, together with the action of the respiratory bellows, determines to a large extent the pressure differential between the atmospheric and intranasal air. The opening, being slightly smaller than the choana, produces, according to Proetz,[10] a negative pressure of minus 6 mm. of water on inspiration and plus 6 mm. of water on expiration. When the aperture is abnormally narrow, the thoracic bellows forces against this constriction and accentuates both the positive and the negative pressure (fig. 7). Too great a positive pressure will cause anemia of the membranes and, because of its obtunding influence on the mucosa, will interfere with the reflexes which inaugurate the mechanism of respiration. An increased negative pressure may result in edema, extravasations and petechiae. When the opening is too large, the thoracic bellows forces against the relatively constricted choana. Intranasal pressures are not developed and the incoming air fails to make a respiratory impression. A subjective feeling of nasal obstruction follows. Proetz [10] stated that the pressure changes in the sinuses are identical with the pressure changes in the nose. Any abnormality in the size of the space between the upper lateral cartilage and the septum must, therefore, result in an interference with drainage and its consequences.

Influence on the Maintenance of Intrathoracic and Intra-abdominal Pressures.—According to the present concept, the nose, aside from being an organ of olfaction, is nothing more than an air conditioner for the lung. While its function of heating, moistening and filtering the inspired air cannot be denied, the importance of these functions has, unfortunately, been overstressed. Too little emphasis has been assigned to its important function of assisting in the regulation of intrathoracic and intra-abdominal pressures. With each respiration the pressures in the thorax and abdomen fluctuate in direct proportion to the depth of the respiration, which, in turn, is controlled, in a measure, by the play of the nasal cartilages. The effect of the negative pressure in the thorax is to force or suck blood from the large veins of the abdomen into the right side of the heart. More blood is thrown, thereby, into the pulmonary circulation and eventually into the left ventricle. This factor would tend to increase the output of the heart during inspiration and raise the arterial pressure.

10. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Pub. Co., 1941.

The descent of the diaphragm on inspiration increases the intra-abdominal pressure, with consequent benefits to the viscera contained therein. Space, however, does not permit a detailed discussion of these benefits.

CAUSATIVE FACTORS

There are many factors which interfere with the normal activity of the regulatory mechanism of the nasal cartilages, and an accurate appraisal of the etiologic fault is essential to proper management.

Faults in Upper Lateral Cartilages.—The upper lateral cartilages may be attached to the septum at too acute an angle, so that even a shallow inspiration may build up sufficient negative pressure to cause

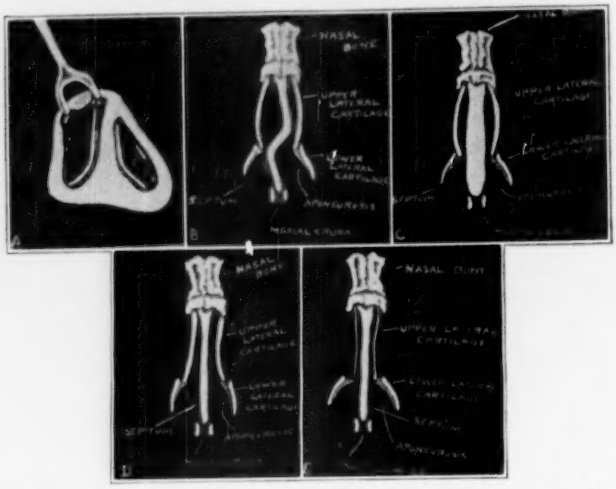

Fig. 8.—Faults in upper lateral cartilages causing collapsed ala. *A*, upper lateral cartilage attached to septum at too acute an angle; *B*, septum buckled or deflected; *C*, septum too thick; *D*, exaggeration of inward curvature of upper lateral cartilages, and *E*, lack of resiliency of upper lateral cartilages.

their collapse. The angle between the septum and upper lateral cartilage may be relatively too acute, owing to buckling, thickening or deflection of the septum or to an undue thickening of the upper lateral cartilage or an exaggeration of its inward curvature. The cartilage itself may lack resiliency and collapse because of its inability to resist the negative pressure (fig. 8).

Faults in Lower Lateral Cartilages.—The lower lateral cartilages play an important role in maintaining the patency of the nasal aperture during forced inspiration. As previously mentioned, they serve as

checkreins on the upper lateral cartilages and prevent them from abutting the septum and shutting off the air current. Too great an inward curvature of the lateral crus, atrophy or paralysis of the alar musculature may interfere with the activity of these cartilages.

Faults in Naral Boundaries.—Obstructions about the nares cause an increased negative pressure above the stenosis, for the air cannot enter rapidly enough to equalize the pressures. This heightened pressure may be sufficient to cause an insuction of the ala. The commonest

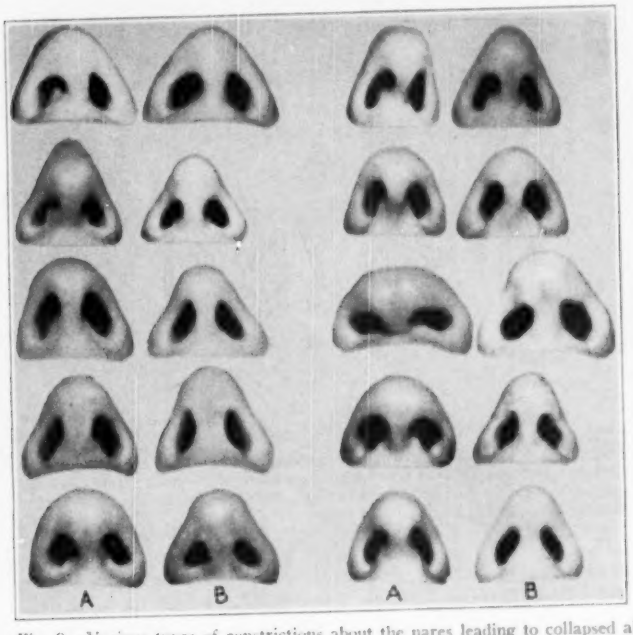

Fig. 9.—Various types of constrictions about the nares leading to collapsed ala. *A*, before operation; *B*, after operation.

conditions which interfere with the entrance of air comprise thickening or buckling of the septum at the nasal inlet; thick columella; thick ala; dislocation of the mesial crus; spurs or ridges on the floor of the nose; elongated, thickened or dislocated nasal spine, or cicatricial contractions (fig. 9).

MANAGEMENT

Up to 1900 the objective in the treatment of alar collapse was to obtain an open airway through some mechanical means. Traube[11]

11. Traube: Report. Berl. klin. Wchnschr. **25**:293, 1871.

suggested the use of a rigid tube or a bent hairpin held to the forehead by adhesive tape. Feldbausch [12] also recommended the use of tubes and invented the elastic wire loops with olive-pointed tips. Jankau,[13] Guye,[14] Ott [15] and Schmidthuisen [16] each perfected a type of tubal dilator. Heermann [17] placed in the vestibular pockets a roll of cotton saturated with boric acid ointment. Menzel [18] suggested injections of paraffin. In recent years, with a better understanding of physiology, together with the incorporation of plastic surgery technics, treatment has been directed toward surgical correction of the causative factor, with a greater assurance of permanent restoration of function than heretofore.

Obviously, since the condition may result from an infinite variety of causes, it would be impossible to describe surgical procedures for the correction of every factor. However, if one familiarizes oneself with the basic operations about to be described, one can easily improvise procedures to meet the specific need.

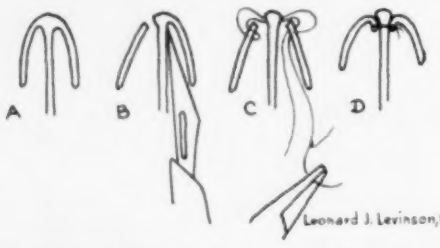

Fig. 10.—Collapsed ala corrected by buckling upper lateral cartilage on septum. *A*, too acute an angle between upper lateral cartilage and septum; *B*, angles between upper lateral cartilage and septum incised; *C*, suture in place to buckle upper lateral cartilages on septum, and *D*, suture tied.

12. Feldbausch: A New Method of Permanent Inhalation of Volatile Substances Through the Nose, Berl. klin. Wchnschr. **17**:672, 1880.

13. Jankau, cited by Weihl, A.: Insuction of the Alae, Ztschr. f. Laryngol. u. Rhinol. **9**:30, 1919-1920.

14. Guye, cited by Weihl, A.: Insuction of the Alae, Ztschr. f. Laryngol. u. Rhinol. **9**:30, 1919-1920.

15. Ott, cited by Weihl, A.: Insuction of the Alae, Ztschr. f. Laryngol. u. Rhinol. **9**:30, 1919-1920.

16. Schmidthuisen, cited by Weihl, A.: Insuction of the Alae, Ztschr. f. Laryngol. u. Rhinol. **9**:30, 1919-1920.

17. Heermann, G.: A Simple Method of Preventing Insuction of the Alae, Ztschr. f. Ohrenh. **56**:165, 1908.

18. Menzel, K. M.: The Treatment of Alar Insuction: A New Method, Munchen. med. Wchnschr. **50**:778, 1903.

FAULTS IN UPPER LATERAL CARTILAGE

In cases in which the angle between the upper lateral cartilage and septum is too acute, it is widened by buckling the upper lateral cartilage on the septum. The following technic is employed (fig. 10): Intra-

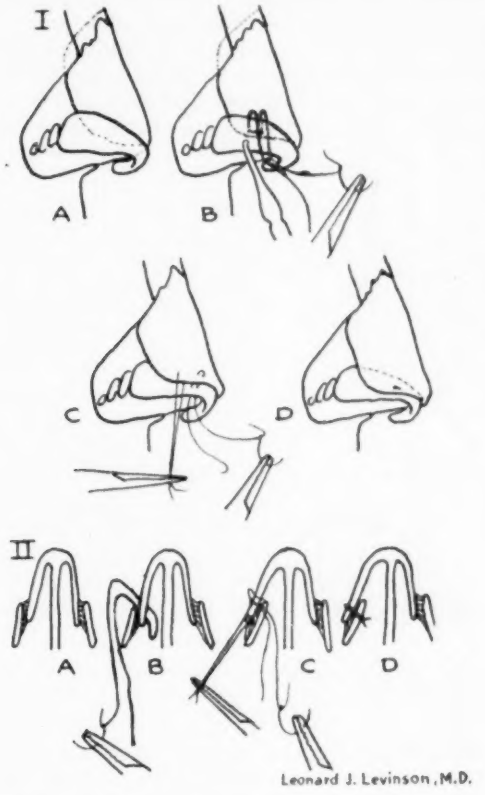

Fig. 11.—Collapsed ala corrected by reversing relation between upper and lower lateral cartilages. I, lateral view; II, cross sectional view. *A*, too acute an angle between upper lateral cartilage and septum; *B*, pilot suture passed through upper lateral cartilage; *C*, upper lateral cartilage drawn over lower lateral cartilage by pilot suture, and *D*, fixation suture in place.

nasal incisions are made along the aponeuroses, connecting the upper and lower lateral cartilages. Through these incisions the soft structures over the nasal dorsum are elevated and the membranous septum cut through as for a typical rhinoplasty. The angles between the upper

lateral cartilages and septum are divided. A mattress suture of 00000 surgical gut is passed through the mucosal surface of one upper lateral cartilage near its cut edge. It is then continued through the septum, the distance from the ventral border depending on the amount of upper lateral buckling desired. The needles are then passed from without in through the upper lateral cartilage on the opposite side, and the suture ends, which now appear in the vestibule, are tied.

If there is an abnormal inward bowing of the cartilage, the relation between the upper and lower lateral cartilages is reversed, the upper lateral cartilages being made to override the lower (fig. 11). Exposure of the upper lateral cartilages and their severance from the septum is

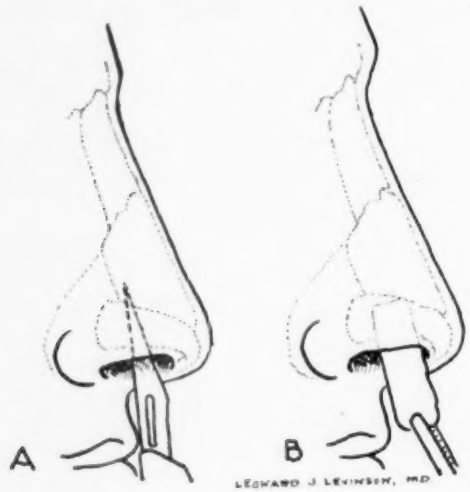

Fig. 12.—Alar collapse due to lack of cartilage resiliency. *A*, soft structures elevated through incision along lower margin of lower lateral cartilage; *B*, section of cartilage implanted.

accomplished as in the previous operation. The lower lateral cartilages are exposed through circumferential incisions along their lower margins. The overlying soft structures are elevated through these incisions, the same technic being used as that for modeling the lower lateral cartilages.[19] The caudal (inferior) end of the upper lateral cartilage is deepithelized for a distance of 5 to 6 mm. A silk pilot suture is passed through the denuded cartilage, carried over the lower lateral cartilage and brought out through the circumferential incision below.

19. Fomon, S.: The Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939.

Traction is exerted on the pilot suture until the upper lateral cartilage overlaps the lower lateral for a distance sufficient to bring about the proper angulation. The cartilage is then fixed in place by a suture passed through the vestibular skin, lower lateral and upper lateral cartilages. The pilot suture is withdrawn. After six or seven days, when adhesions will have taken place, the fixation suture is removed.

When either of these conditions occurs in a nose which is also unduly long, the standard technic employed for shortening the nose may accomplish the double purpose of correcting the cosmetic deformity and, at the same time, widening the angle.[19]

If the angle between the upper lateral cartilage and septum is relatively too acute because of buckling, thickening or deflection of the

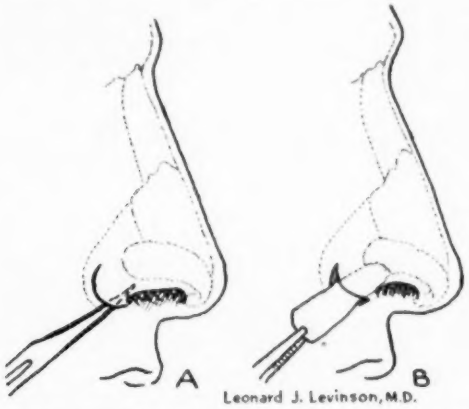

Fig. 13.—Cartilage graft introduced through opening along alar-facial groove (Schattner [21]).

septum, a submucous resection is performed, after which a straight piece of cartilage is inserted between the membranes. Failure to replace the cartilage may lead to an aggravation of the collapse, for the negative pressure during inspiration may cause the loose membrane to flap against the upper lateral cartilages.[20]

If the upper lateral cartilages are unduly thickened, they are exposed and separated from the septum in the usual manner. The cartilage is steadied with a dural hook and shaved down with a no. 11 Bard Parker knife to the required thinness.

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20. Fomon, S.: Syracuse, V. R.; Bolotow, N., and Pullen, E. M.: Plastic Repair of the Deflected Nasal Septum, Arch. Otolaryng. **44**:141 (Aug.) 1946. Fomon, S.; Gilbert, J. G.; Silver, A. G., and Syracuse, V. R.: Plastic Repair of the Obstructing Nasal Septum, ibid. **47**:7 (Jan.) 1948.

When the fault lies in the lack of resiliency of the upper lateral cartilage, the latter cartilage may be stiffened by the implantation of a shaped cartilage graft in the following manner (fig. 12): An incision is made in the vestibule along the lower margin of the lower lateral cartilage. The soft structures are elevated in the plane above the perichondrium to a point just above the nasal bone. A measured section of cartilage removed from the septum or a piece of

Fig. 14.—Correction of collapsed ala caused by inward bulging of lateral crus. *A*, incision made in vestibule along caudal margin of bowed cartilage; *B*, lateral crus delivered and cross hatched, and *C*, vestibular skin attached to outside skin by perforating suture.

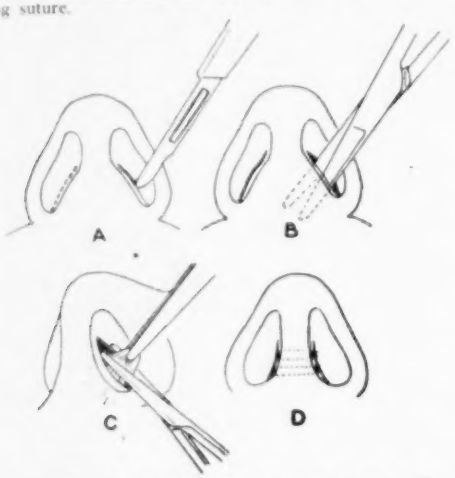

Fig. 15.—Correction of collapsed alae due to thick columella. *A*, incisions made along cephalic margins of mesial crura; *B*, columellar skin separated from underlying structures; *C*, excess tissue removed, and *D*, mesial crura united with mattress sutures of black silk.

pickled septal cartilage is shaped and introduced into the prepared pocket. The graft is held in place with intranasal packing, supported externally by a stent dressing. Schattner [21] preferred to introduce the graft through an opening made along the alar facial groove (fig. 13). If properly shaped, the implant produces no external deformity.

21. Schattner, A.: Personal communication to the authors.

FAULTS IN LOWER LATERAL CARTILAGE

If the lateral crus bows inward, the management will depend on whether the bowing is partial or complete. If only the cephalic margin bulges inward, so as to impinge on the septum at inspiration, an incision is made in the vestibule along the caudal margin of the bowed cartilage. The vestibular skin is dissected away from the cartilage, which is then cross hatched or removed. By means of a triangular perforating suture tied on the vestibular surface, as shown in figure 14, the cartilage is bowed outward. If the entire lateral crus bows inward, an incision is made along its caudal border. The outer and the vestibular skin are separated from the cartilage, which is then removed, reversed and reinserted, convex surface out.

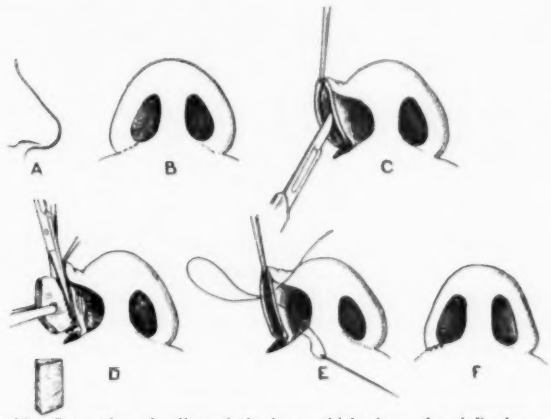

Fig. 16.—Correction of collapsed ala due to thick alae. *A* and *B*, alae separated from cheeks along alar-facial grooves; *C*, sharp dissection of fatty pad between skin layers; *D*, resection of fatty wedge; *E*, edges of skin brought together with silk suture so passed that loop disappears beneath cutaneous surface, and *F*, alae reattached.

If the cartilage lacks resiliency, the alar wall is stiffened by the implantation of a thin plate of cartilage.

CONTRIBUTING CAUSES

If the columella is too thick because of excessive subcutaneous tissue or because of too much flaring of the extremities of the mesial crura, an incision is made along the cephalic (superior) margin of the mesial crus and the excess subcutaneous tissue or cartilage is caught with a rat-tooth forceps and cut away with a Stevens scissors. If a hypertrophied or dislocated spine is responsible for the thickened columella, it is chiseled off. A through and through mattress suture of silk is

passed through the columella walls to hold them in close approximation. The initial incision is closed with two or three atraumatic sutures (fig. 15).

If the alae are so thick that they impinge on the naral opening, correction is accomplished in the following manner (fig. 16): The ala is separated from the cheek along the alar-facial groove. The cut edge shows a definitely demarcated fatty pad between the vestibular and external skin. This fatty wedge is removed with a no. 11 Bard Parker knife, and the skin surfaces are brought together with a silk suture. The suture is so passed that it is returned through the same skin opening through which it was brought out and tied in the vestibule, thus obviating any danger of surface scarring.

If the caudal (inferior) end of the septum is subluxated, an incision is made along the deflected margin, the membranes are elevated on both sides, and the cartilage is removed. If it can be straightened by cross hatching or thinning, it is replaced; otherwise, a piece of pickled cartilage is used.

When scar tissue constricts the nostril, it is excised and the raw surface covered with a split skin graft taken from the thigh. A Kyle tube is worn for the first month or two to lessen subsequent contraction.

SUMMARY

The syndrome of collapsed ala is discussed from the standpoint of its pathologic physiology. The importance of the nasal cartilages in lung ventilation, mixing of air, regulation of rhythm and intranasal, intrathoracic and intra-abdominal pressures is emphasized. The causative factors are enumerated and the surgical treatment outlined for their correction.

BONE CONDUCTION IN OTOSCLEROSIS

R. R. WOODS, M.D.
DUBLIN, EIRE

OTOSCLEROSIS has always been described as typical conduction deafness. The reason for this description is that until recent years Rinne's and Schwabach's tests, on which the diagnosis between conduction and perceptive deafness depends, were always performed with a low-pitched tuning fork, usually middle C (256 vibrations per second); and in otosclerosis bone conduction at this pitch remains normal for a very long time, even when it is grossly reduced at 512 cycles per second and higher frequencies.

For the past fifteen years routine tests have been performed in all cases with the air and bone conduction audiometer, and it has been found that except in relatively early cases bone conduction is seldom normal in the upper frequencies. As soon as the hearing falls below a certain level bone conduction in the higher frequencies begins to drop down, so that as one watches a case of otosclerosis develop one has the impression of a disease which produces a middle ear type of deafness in the low tones and a mixed deafness in the upper tones. In advanced cases bone conduction is reduced in the low tones also; and, if it is assumed that bone conduction is indicative of nerve function, it must be concluded that otosclerosis produces from the first a mixed deafness, the conductive element of which preponderates in the early stages, the perceptive element becoming more pronounced as the disease advances and finally becoming much the more important of the two. In very advanced cases the conductive element seems to account for relatively little of the deafness, which is at this stage mainly due to the perceptive element. So much is this so, that there can be some difficulty in making a differential diagnosis between very advanced otosclerosis and pure perceptive deafness. Thus the disease starts as pure conduction deafness, then passes through a mixed stage, and the further it advances, the closer it approximates perceptive deafness. Again, as one watches the condition progressing, two apparently opposed facts stand out: In the early stages, hearing by air conduction is reduced in the low tones while remaining normal for the high tones; later, when bone conduction begins to be affected, hearing by bone conduction is reduced in the high tones while remaining normal for the low tones. In other words, the earliest air conduction loss is in the low tones, while the earliest bone conduction loss is in the high tones.

In this study the Western Electric 2 B audiometer* has been used, with the D-80904 bone conduction receiver. A word must be said about bone conduction testing. The bone conduction receiver is held in the examiner's hand, not the patient's. In this way the receiver can be used as if it were a tuning fork emitting a constant intensity, which is immediately variable at will. No claim is made for the absolute accuracy of bone conduction tests; nor is it at all certain that the decibel potentiometer which is used with the air conduction receiver is equally applicable to the bone conduction receiver.[1] But the least that can be said of the bone conduction audiometer is that it is enormously more accurate than tuning forks. With the bone conduction audiometer one can at least produce a note of constant intensity and repeat this constant intensity again at will. Testing with the tuning fork and the stopwatch is open to errors so enormous as to preclude their use in any but a qualitative way. To test hearing with a note of constantly diminishing intensity is analogous to testing sight with letters which are gradually diminishing in size and asking the patient to say at what point they become too small for him to read. The bone conduction receiver should be regarded as a mechanical tuning fork. When used in this way, it is quite consistent with itself, and repeat tests give results which are as consistent as the results of air conduction audiometry. It is quite possible that the prejudice which is so frequently evident against audiometry in general and bone conduction audiometry in particular is due to the relegation of audiometric testing to nurses or audiometrists. All audiometry ought to be carried out, or at least checked, by the otologist himself. No audiometrist has been employed in the series of cases reported here, and all tests have been checked and rechecked to insure accuracy. In any case, for the purpose of this communication, the absolute accuracy of bone conduction readings is not of prime importance; what matters is the relative levels of preoperative and postoperative readings.

In all cases bone conduction has been tested with the opposite ear masked (masked bone conduction). The masker used was an electric buzzer mounted in an ear piece made from a covering for a colostomy stoma, the rubber mount of which fits closely to the skull. This gives a masking noise of 70 to 80 decibels above threshold. A certain amount of noise escapes so that the tested ear as well as the untested ear is masked to some extent. This appears to be inevitable, and as a result the level of masked bone conduction is usually slightly below that of unmasked bone conduction. This reduction averages 5 to 10 decibels, unless there is some concealed loss of bone conduction which has not been demonstrated by the unmasked bone conduction test. The spectrum of this masker has not been analyzed, but it has been found that in cases of complete unilateral nerve deafness, when the normal ear is

1. Tumarkin, A.: What We Can Deduce from Quantitative Estimations of Bone Conduction, J. Laryng. & Otol. **61**:473, 1946.

masked by it, all bone conduction is abolished from the deaf ear except at the pitch of 64 and 128, at which bone sensation is too potent a factor for the test to be of any value. These frequencies are not now used in testing bone conduction.

It has been stated that the fenestration operation has no effect on the acuity of hearing by bone conduction. In fact, this has been stated so often that it has come to be accepted as a fact without being questioned. It is theoretically what should be expected, for otosclerosis has always been described as a typical conduction deafness; and although of recent years we physicians have been forced to realize that in many cases there are changes in the perceptive mechanism also, we have described these changes rather vaguely as "secondary cochlear degeneration" or "secondary nerve degeneration," and we have regarded such degeneration as analogous to, and probably due to the same sort of causes as, the degenerations of pure nerve deafness. In other words, we have assumed, unconsciously perhaps, that the perceptive element in otosclerotic deafness once firmly established was irreversible.

This, more or less, was the point of view that I had when I first started performing the fenestration operation. Routine postoperative testing, however, soon demonstrated that in the vast majority of cases the acuity of hearing for bone conduction following fenestration showed a definite gain over the preoperative level. A preliminary report on this was published in 1948.[2] A consecutive series of cases have now been investigated from this point of view, and the results are analyzed here.

At the outset, it seemed clear that one could expect improvement in bone conduction hearing in only those cases which showed an improvement in air conduction hearing, and that therefore all cases which showed no air conduction improvement following operation must be excluded. Exclusion of these cases leaves a total of 46 cases to be reviewed here.

Now it also seemed clear that one could not expect improvement in bone conduction hearing unless the preoperative bone conduction was reduced below normal. In a case with normal bone conduction, better than normal bone conduction was unlikely to develop. What then can be regarded as normal bone conduction? It was decided to regard as normal a masked bone conduction in which the average loss for the three conversation frequencies did not drop below 10 decibels. This would not appear to be too liberal an allowance for normality, especially when it is remembered that the untested ear is masked. This always lowers the bone conduction level in the tested ear by about 5 decibels and occasionally more. There were 7 cases in which masked bone conduction was normal, as judged by this standard (chart 1). This leaves 39 cases in which the preoperative masked bone conduction was

2. Woods, R. R.: Some Observations on Bone Conduction Following the Fenestration Operation, J. Laryng. & Otol. **62**:22, 1948.

reduced and in which hearing was improved. In these cases, after the fenestration operation, the average decibel gain for masked bone conduction in the conversation frequencies was 11.4 decibels. The ears not operated on have been used as controls, and the corresponding figure for them is an average gain of 1.1 decibels. This method of averaging the gains over a whole series of cases and controlling the results by comparison with the ear not operated on eliminates errors on the part of both examiner and patient and produces a figure which is really significant. It is clear that if 39 cases showed an average increase of 11 decibels in bone conduction hearing following operation, the statement that the fenestration operation has no effect on bone con-

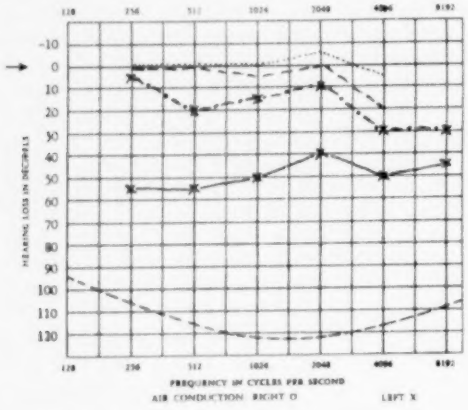

Chart 1 (group 1).—An example of normal preoperative bone conduction; as expected, postoperative bone conduction shows no appreciable change. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by crosses, dots and dashes, and postoperative masked bone conduction, by dots.

duction cannot be true, and the whole question deserves reconsideration and reinvestigation. Of course 11 decibels is an average figure; in some cases there were greater and in some smaller gains than this.

The distribution of cases with regard to the amount of gain is shown in chart 2. There is a central group of 32 cases in which the masked bone conduction showed an increase of between 7 and 15 decibels. There is a group of 4 cases in which no change occurred, for an increase of 5 decibels or less is too near the experimental error of testing to be of any significance. There is a third group of 3 cases in which increases greater than 15 decibels were observed. Thus out of 39 cases, 35 (or practically 90 per cent) showed a significant increase in bone conduction, while in only 10 per cent there was no change. It is noteworthy that in no case was any loss shown.

The group of 4 cases in which there was no change merits further consideration. One of the patients of this group had two separate attacks of postoperative labyrinthitis; the first occurred about a week after operation when after an apparently calm postoperative period, third degree nystagmus developed toward the side operated on and persisted for four or five days. The patient's hearing at the first postoperative test was very much below the preoperative level, but during the next five weeks it gradually improved to give her quite good hearing. Two months after operation she had a second similar attack with nystagmus and dizziness, and her hearing dropped to a very low level again. It subsequently recovered but never came quite up to its best level. These labyrinthine attacks, whatever their origin, may easily have been responsible for the patient's failure to show any improvement in bone

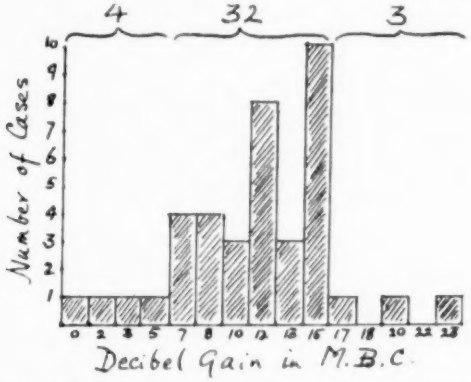

Chart 2.—Distribution of cases with respect to amount of gain in masked bone conduction. Each figure is carried out to the nearest whole decibel, and as each is an average of three, there are certain figures missing. There is a central group of 32 cases with a gain of between 7 and 15 decibels; in 3 cases there are gains of more than this, and in 4 cases there is no significant change (average for 512, 1048 and 2048 cycles per second).

conduction. It is difficult to account for the other 3 patients except to say that their improvement in hearing, although satisfactory, never reached quite as high a level as might have been expected. It is possible that there was some operative trauma to the labyrinth which prevented bone conduction from improving and limited the improvement in air conduction. It may be that measurement of postoperative bone conduction may give some indication of labyrinthine damage, not by showing a loss but by showing a failure to gain.

The central group in which was shown an increase of 7 to 15 decibels is by far the largest, comprising 32 of the 39 cases. Chart 3 shows a case with a small but definite improvement of bone conduction hearing

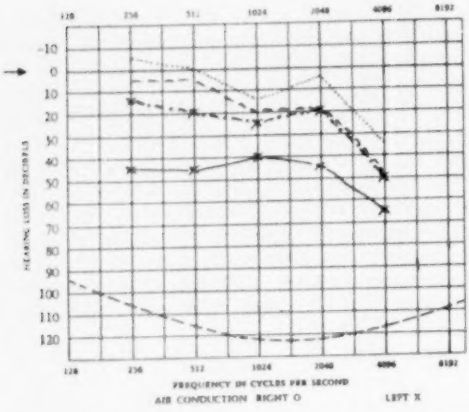

Chart 3 (group 2).—Preoperative hearing loss for masked bone conduction averages 15 decibels for the conversation frequencies; postoperative hearing loss is 7 decibels. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by crosses, dots and dashes, and postoperative masked bone conduction, by dots.

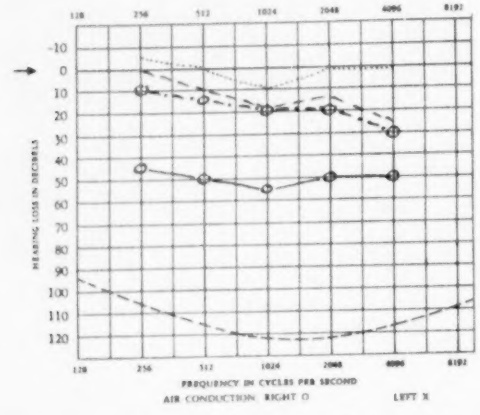

Chart 4 (group 2).—Preoperative hearing loss for masked bone conduction averages 15 decibels for the conversation frequencies; postoperative audiogram shows an average masked bone conduction gain of 12 decibels. Air conduction hearing following operation has risen to near the preoperative masked bone conduction level. Preoperative air conduction is indicated by a solid line and circles; preoperative masked bone conduction, by a broken line; postoperative air conduction, by circles, dots and dashes, and postoperative masked bone conduction, by dots.

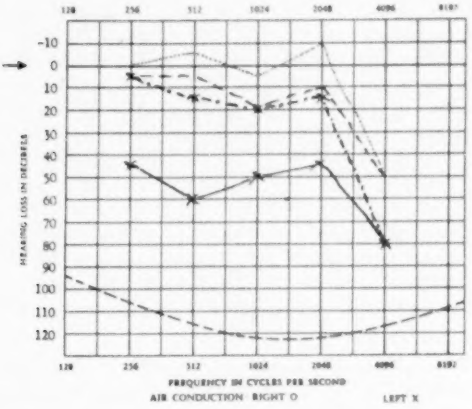

Chart 5 (group 2).—Preoperative hearing loss for masked bone conduction averages 12 decibels for the conversation frequencies; the postoperative audiogram shows an average masked bone conduction gain of 15 decibels. Air conduction hearing following operation has risen to near the preoperative masked bone conduction level. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by a line of crosses, dots and dashes, and postoperative masked bone conduction, by dots.

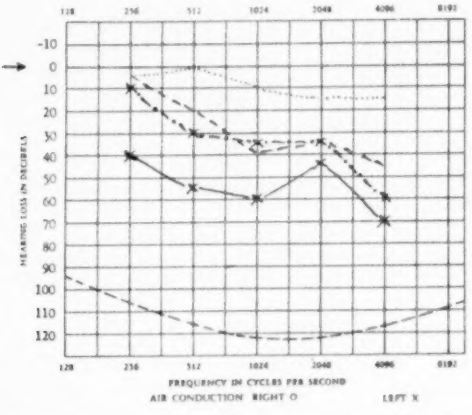

Chart 6 (group 4).—Preoperative hearing loss for masked bone conduction averages 32 decibels; the postoperative audiogram shows an average gain in masked bone conduction of 23 decibels. The bone conduction gain is greater than the air conduction gain. Preoperative and postoperative tests were repeated frequently. The audiogram of the ear not operated on is shown in chart 7. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by a line of crosses, dots and dashes, and postoperative masked bone conduction, by dots.

(8 decibels). This sort of small improvement is usually more noticeable in the high frequencies than in the low. Chart 4 shows a case with a 12 decibel increase, and chart 5 shows one with a 15 decibel increase.

Finally, there are the 3 cases with increases of more than the average. Chart 6 shows the most pronounced of these with an average bone conduction gain of 23 decibels, compared with an average air conduction gain of 20 decibels. The obvious suggestion is that this is an error, but chart 7 shows the ear not operated on in which there has been no change in bone conduction. Furthermore, there was close agreement between repeated audiograms. Four preoperative masked bone conduction tests were done, and the postoperative bone conduction curve was frequently checked. The patient was very intelligent and answered tests well and the possibility of gross error can be eliminated.

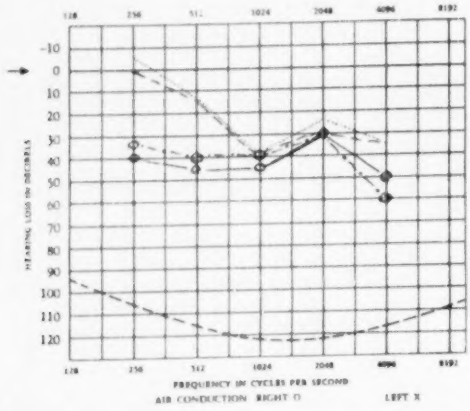

Chart 7.—Audiogram of ear not operated on in case shown in chart 6. There is no significant change in air or masked bone conduction. This case shows how closely otosclerosis can at times approximate to a perceptive type of deafness. Preoperative air conduction is indicated by a solid line and circles; preoperative masked bone conduction, by a broken line; postoperative air conduction, by circles, dots and dashes, and postoperative masked bone conduction, by dots.

It is of interest to study these figures not only by themselves but on the basis of the preoperative loss of bone conduction to see if gain in postoperative bone conduction hearing bears any relation to preoperative bone conduction loss. This is shown in table 1, in which the 7 cases with normal preoperative bone conduction have been included for comparison. Here cases have been classified according to the average preoperative bone conduction loss in the conversation frequencies into groups in steps of 10 decibels, and the averages are shown for each group. Group 1 comprises normal cases; group 2, cases with

10 to 20 decibel loss; group 3, cases with a 20 to 30 decibel loss, and so on. This table shows many points of interest. In the first place, the postoperative tests of the ears not operated on show no material change. These tests act as controls. Operation produced no material change in the cases with normal preoperative bone conduction (group 1), the average gain of 1.9 decibel in the ears operated on and the average loss of 1.7 decibel in the ears not operated on being quite insignificant. This result is what was expected. The figures for group 5 (40 decibel loss and more) are of no significance, as there were only 2 cases in the group. In groups 2, 3, and 4 there appears to be progressive increase in the bone conduction gain; the lower the preoperative bone conduction, the greater the gain following operation. The numbers, however, are too small and the differences not large enough to be very convincing, but there appears to be a tendency toward this direction. It is interesting to note that the postoperative loss in bone conduction in group 2 is the same as in group 1; in other words, the bone conduction in group 2 has been restored to normal. As a matter of fact, in each group opera-

TABLE 1.—*Data on Air Conduction and Masked Bone Conduction Before and After Operation*

		Masked Bone Conduction				Air Conduction	
		Ears Operated On			Ears Not Operated On		
Group	Number of Cases	Preoperative Loss, Decibels	Average Post-operative Loss	Average Gain	Change in M. B. C.	Average Post-operative Loss	Average Gain
1	7	0-10	4.7	1.9	—1.7	17.8	29.1
2	22	10-20	3.9	10.9	+1.5	23.6	26.3
3	10	20-30	13.8	11.2	+0.5	31.1	22.8
4	5	30-40	19	15.5	+2.7	46.6	19.6
5	2	40—	35	8	+1	68.5	17.5

tion has raised the average level of bone conduction to that of the preoperative level of the group previously mentioned.

Table 1 also shows the average decibel gain for air conduction and the final postoperative average decibel loss. As the preoperative bone conduction level drops, there is a progressive fall in the average decibel gain for air conduction. This is instructive because it shows that although one may expect an average increase of 30 decibels when the bone conduction is normal, one cannot expect as high an increase as this if the bone conduction is reduced even 15 to 20 decibels, a level which is generally regarded as fairly favorable. This is not to say that high gains cannot be obtained when bone conduction lies at such a level, but the average will be lower, and it is on the average that prognosis must be based. With small losses in bone conduction the postoperative air conduction audiogram can be pushed up to somewhere near the preoperative bone conduction line, and the gap between preoperative bone conduction and air conduction is closed (charts 4 and 5). With greater degrees of bone conduction loss this gap is rarely closed, and

the postoperative air conduction level does not usually approach the preoperative bone conduction level (chart 11). This seems to confirm the opinion expressed earlier in this paper that the conductive element in otosclerosis becomes of diminishing importance as the disease advances. Similarly, the level to which the postoperative hearing rises diminishes as the preoperative bone conduction loss increases. Group 1 shows an average postoperative loss for air conduction of 17.8 decibels, and group 2 an average of 23.6 decibels, both well within the practical limits; group 3, with an average of 31.4, is just outside the practical level, though in many cases in this group the practical level was reached.

There is one further point to be noted. In group 2 the increase in bone conduction (10.9 decibels) is 40 per cent of the increase in air conduction (26.3 decibels). In group 3 it is 50 per cent, while in group 4 the increase in bone conduction (15.5 decibels) is more than 75 per cent of the increase in air conduction (19.6 decibels). The increase in bone conduction in relation to the increase in air conduction is relatively much greater in the lower groups than in the higher. Also, in the lower groups the increase in bone conduction is actually greater, while the increase in air conduction is less than in the higher groups.

These, then, are the facts as observed. There seems no reason to doubt that, since the improvement in bone conduction, like the improvement in air conduction, follows immediately on fenestration of the labyrinth, the alteration in both must be attributed to the operation. It might be suggested that improvement in bone conduction could be due to alteration of bone conductivity consequent on the exenteration of the mastoid cells, but bone conduction is not altered when the mastoid cells are exenterated without fenestration, as in radical mastoidectomy. Furthermore, improvement in bone conduction disappears when the fenestra closes (charts 8 and 9). Such cases are fortunately not common, and this was the only one that occurred in this series. It illustrates the point, though not very dramatically. Three months after operation, the hearing lay above practical level, though the degree of improvement was hardly satisfactory (18 decibels) and the bone conduction hearing showed an average increase of 8 decibels (chart 8). Six months after operation the fenestra had closed, the fistula symptom was negative and both air and bone conduction had returned to about the preoperative level (chart 9).

It must therefore be concluded that the improvement in bone conduction is due to the fenestration of the labyrinth and that it occurs in the majority of cases (90 per cent in this series). Further evidence in support of this conclusion is an observation of Shambaugh's that when the fenestra stays open and hearing remains good, nerve degeneration does not progress; but when the fenestra closes, nerve degeneration proceeds as before.

These results are unexpected. Theoretically, one would have expected a successful operation to raise the level of hearing by air conduction to the preoperative bone conduction level or thereabouts, so that the postoperative audiogram would have given the picture of an internal ear deafness, a preoperative mixed deafness with the middle ear element abolished, or nearly so. Instead of this, the postoperative audiogram still preserves all the characteristics of a mixed deafness, both elements of which have been alleviated. In fact, the postoperative audiogram resembles that of an early stage of the disease.

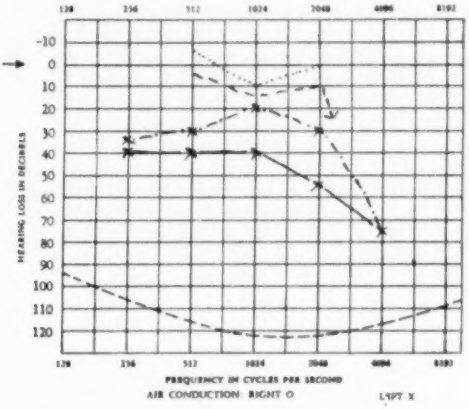

Chart 8.—Audiogram taken three months after operation; masked bone conduction shows an average gain of 8 decibels. The audiogram taken six months after operation is shown in chart 9. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by crosses, dots and dashes, and postoperative masked bone conduction, by dots.

It would therefore appear evident that at least part of the loss of bone conduction in otosclerosis is due to a change which is reversible and cannot therefore be due to degeneration of the organ of Corti or the cells of the spiral ganglion, or, in fact, to any of the usual causes of "nerve deafness." This part of the bone conduction loss cannot be attributed, as has hitherto been usual, to "secondary cochlear degeneration." Now in early cases with slight or moderate degrees of bone conduction loss (say an average of 10 to 15 decibels) the whole of the bone conduction loss is reversible. After operation, bone conduction becomes normal, and a case which shows an audiogram of mixed deafness before operation, after operation presents pure conduction deafness, the slight hearing loss which remains being probably due to the disorganization of the middle ear structures at operation. The ear seems

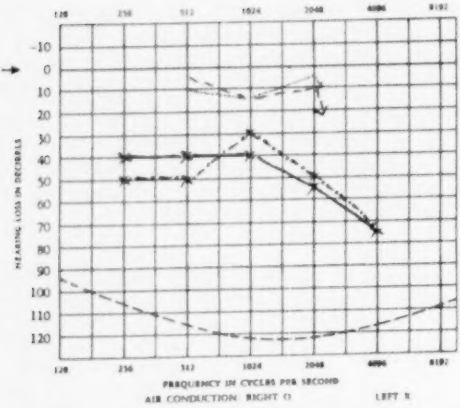

Chart 9.—Audiogram taken six months after operation in same case as in chart 8. The fenestra has closed and no fistula symptom can be elicited. Bone conduction has returned to the preoperative level. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by crosses, dots and dashes, and postoperative masked bone conduction, by dots.

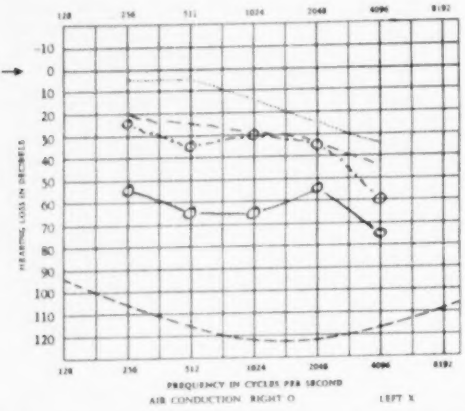

Chart 10.—Preoperative masked bone conduction hearing is much reduced (average loss, 30 decibels). The postoperative masked bone conduction, though improved, does not nearly reach normal, and the postoperative audiogram still presents the picture of mixed deafness, as might have been the case some years previously. Preoperative air conduction is indicated by a solid line and circles; preoperative masked bone conduction, by a broken line; postoperative air conduction, by circles, dots and dashes, and postoperative masked bone conduction, by dots.

to have been put back to the condition in which it was some years previously. Charts 1, 3, 4 and 5 illustrate this. In later cases (chart 10) bone conduction, although improved, does not reach normal, and the case still appears as mixed deafness, as it might have been at an earlier stage of the disease. This is even more obvious in severely deafened patients, as is shown in chart 11. In early cases, then, loss of bone conduction is completely reversible; in later cases, it is only partially reversible, and one may be permitted to argue in the opposite direction that otosclerosis produces a change in the ear that causes loss of bone conduction which is at first reversible but later becomes permanent, and that by the time this happens further reversible changes have developed.

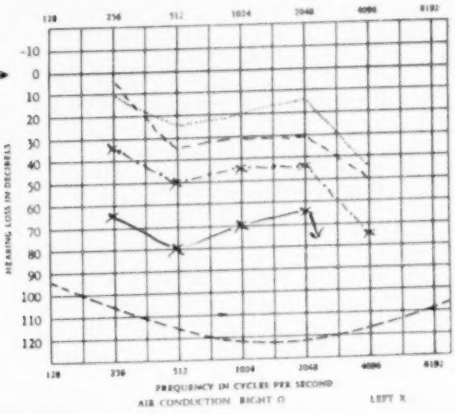

Chart 11.—Audiogram in a case of severe deafness, with considerable loss of bone conduction. Postoperative air conduction does not rise to the preoperative bone conduction level. After operation the case still appeared as one of mixed deafness. Preoperative air conduction is indicated by a solid line and crosses; preoperative masked bone conduction, by a broken line; postoperative air conduction, by crosses, dots and dashes, and postoperative masked bone conduction, by dots.

There is a limit to the degree of deafness that can be caused by stapes fixation alone. If in a normal patient a complete stapes fixation were to develop suddenly, he would have deafness of a certain degree, and not more. Any further degeneration in hearing would have to be due to other causes—presumably failure of perception. It is a question how much deafness stapes fixation alone can produce, but it would seem to be in the neighborhood of a 50 to 55 decibel loss at 512 cycles per second and a 40 to 45 decibel loss at 2048 cycles per second. In group 1, with normal bone conduction, the average air conduction loss was 53 decibels at 512 cycles per second and 41 decibels at 2048 cycles per second. Whereas for the group with a bone conduction loss of

11 to 15 decibels the average air conduction loss was 55 decibels at 512 cycles per second and 43 decibels at 2048 cycles per second (table 2). This would seem to be about the average level of air conduction hearing when loss of bone conduction first starts to appear. It must be admitted, however, that this is rather variable from case to case; sometimes bone conduction remains normal until stapes fixation is complete and sometimes it starts to drop earlier in the disease. In this series the earliest it has occurred was in a case in which air conduction hearing stood at 35 decibel loss for each of the conversation frequencies.

Increase in acuity of hearing for bone conduction following the fenestration operation seems to be evidence that stapes fixation per se causes a loss in bone conduction, and that otosclerosis causes, therefore, from the start a mixed deafness or at least a potentially mixed deafness. The cause of the reduced bone conduction is another question, but it seems reasonable to suppose that fixation of the stapes not only prevents vibrations from reaching the labyrinth but also impedes the response of the end organ by limiting the mobility of the perilymph. If this were so, the creation of a fenestra, in addition to short-circuiting the middle

TABLE 2.— *Relation of Loss of Air Conduction to Loss of Bone Conduction*

Group	Loss of Masked Bone Conduction, Decibels	Average Air Conduction Loss at 512 Cycles per Second	Average Air Conduction Loss at 2048 Cycles per Second
Normal bone conduction	0-10	58	41
Impaired bone conduction	11-15	55	43

ear block, would also free the perilymph and allow it to vibrate and stimulate the organ of Corti, thereby alleviating both the middle and the inner ear elements. This sort of explanation, expressed mathematically, might explain why the earliest air conduction defect is in the low tones, whereas the earliest bone conduction defect occurs in the high tones. Such a hypothesis would explain the early cases in which the loss of bone conduction is wholly reversible; but in the later cases, in which it is only partially reversible, there must clearly be another factor involved. This factor may be again a direct consequence of stapes fixation, such as an atrophy of disuse or it may have a totally different origin. It is noteworthy that all forms of middle ear deafness tend to develop perception deafness, as evidenced by loss of bone conduction, in their later stages. At any rate, it is clear that although bone conduction may be an indicator of the stimulability of the cochlea under the conditions of stapes fixation, its value as an indicator of the potentially maximum cochlear function is obviously extremely doubtful.

However interesting this phenomenon may be from the physiologic point of view it is of even greater importance for the practicing otologist, for he relies on the measurement of bone conduction to enable him to select his cases for fenestration and to give a prognosis as to the prob-

able result of operation. It is therefore essential for him to have a clear knowledge of the mechanism of bone conduction in order to be able to arrive at an accurate clinical interpretation of the test.

SUMMARY

Acuity of hearing for masked bone conduction before and after the fenestration operation has been investigated in a consecutive series of cases. Bone conduction in the ears operated on showed an average improvement of 11.4 decibels. The ears not operated on were used as controls and showed no significant change. Ninety per cent of the cases showed improvement of masked bone conduction following operation. Part, at least, therefore, of the loss of bone conduction in otosclerosis is reversible and cannot be attributed to atrophy of disuse or secondary nerve degeneration, or to any other condition which would produce an irreversible change. The results are analyzed and the question of loss of bone conduction in otosclerosis is discussed. In otosclerosis, the first air conduction loss is in the low tones, while the first bone conduction loss shows itself in the high tones. In early cases both these losses are reversible by operation, the remaining air conduction loss being due to disorganization of the middle ear. In later cases neither air nor bone conduction losses are wholly reversible. The postoperative audiogram resembles that of an earlier stage of the disease. The opinion is expressed that stapes fixation, per se, causes a loss in bone conduction and that otosclerosis produces from the start a mixed, or at least a potentially mixed, deafness, the conductive element of which is predominant in the early stages, the perceptive element becoming more and more important as the disease advances.

39 Merion Square.

SOME EFFECTS OF THE COMMON COLD ON SPEECH

CHARLES LIGHTFOOT, Ph.D.
LOS ANGELES

FREQUENTLY heard are remarks which imply that when one has a cold one's speech sounds "different." This commonly alleged difference has evidently been so generally accepted that scientific investigators have not been stimulated to evaluate it; at least, a search that I made through the literature revealed few observations other than what might be furnished by any man on the street. I felt that some attempt should be made to obtain more definite information about the influence a cold might have on speech not only because of the frequency of opportunities that any such relationship has to exert itself, but also because information concerning it might add to the understanding of other voice problems. By way of initiating such an attempt, a number of victims of coryza were found and several possible effects of that condition on their speech were studied. The purpose of this paper is to report findings of that study.

Specific questions posed for investigation were the following: 1. To what extent does having a cold affect a speaker's intelligibility or vocal quality? 2. If a cold modifies speech, does the effect vary with the different acoustic conditions under which the listeners respond? 3. Does nasal spraying with a vasoconstricting agent influence the relations implied by the preceding questions? 4. Are such relations associated with medical symptoms which characterize a cold or with phonemes which require nasal emission of sounds?

PROCEDURE

Twenty-four subjects were selected from a group of aviation cadets and enlisted men at the United States Naval Air Station, Pensacola, Fla. Every one in the group had been referred by physicians in the station dispensary after having been diagnosed as having a cold. No one referred in a febrile condition or with sinusitis, tonsillitis, laryngitis, bronchitis or otitis media was used in the investi-

The research reported here was done in the Acoustic Laboratory of the United States Navy School of Aviation Medicine, Pensacola, Fla., under contract N7onr-411 between Special Devices Center of the Office of Naval Research and Kenyon College. Part of this paper, which constitutes ONR Technical Report SDC-411-1-8, was read at the annual conference of the Central States Speech Association, Omaha, April 30, 1949.

gation, and no one was used if his audiogram showed a hearing loss greater than 20 decibels for any frequency between 256 and 8192 cycles or an average loss greater than 10 decibels. In other words, the subjects were troubled primarily by simple coryza uncomplicated by symptoms other than acute rhinitis and/or nasopharyngitis.

The procedure followed for each subject involved obtaining an evaluation of the severity of his coryzal symptoms,[1] a test of his auditory acuity and two high fidelity phonograph records of his speech. The first record represented the subject's reading of a twenty-four word intelligibility test list [2] before he had received any treatment for his cold; the second record, his reading of a comparable list five minutes after a nasal spray of phenylephrine hydrochloride (neosynephrine hydrochloride®)—0.25 per cent solution—had been administered by a physician. The subject was then sent back to the dispensary for further treatment. In a week or ten days, after a second medical examination indicated that the coryzal symptoms had disappeared, he returned to the laboratory and a third record was made, representing his reading of a list in his normal voice. The subject was then dismissed.

The rest of the experimental procedure consisted in getting reactions of listeners to various aspects of the recorded utterances played back under various conditions. Details of the several types of listener response will be given with the presentation of the results.

RESULTS

Intelligibility.—Mean percentage intelligibility scores representing six combinations of experimental conditions and the standard deviations of their six distributions are represented in chart 1. The three records of each subject were each presented to two panels of nine to eleven listeners. One panel, seated in a large classroom, identified a subject's words as they were transmitted at a rather low level through a high fidelity loudspeaker 20 feet (7 meters) away. Speaker intelligibility means derived from such identifications are represented by the open bars in the figure.

Another panel, seated in a smaller room saturated with airplane-like noise (105 decibels above threshold), identified the same subject's words as they were transmitted through headphones characterized by high frequency attenuation. Means derived from such identifications are represented by the striped bars. The first pair of bars represent the first recording conditions, i. e., coryzal speech; the second pair represent treated-coryzal speech; the third pair, the third recording condition, normal speech.

The fact that the open bars in the figure are all slightly shorter than the striped ones need cause no concern, since it was not the purpose of the experiment to compare the intelligibility associated with the two playback conditions. The question of interest concerning playback

1. Dr. Calvin J. Curts (MC) U.S.N. furnished the evaluations.

2. Haagen, C. H.: Intelligibility Measurement: Twenty-Four-Word Multiple-Choice Tests, report no. 5567, PB no. 12050, Office of Scientific Research and Development, United States Emergency Management Office, Sept. 11, 1945. PB No. 12050 United States Emergency Management Office, Sept. 11, 1945.

conditions was whether or not their effects influenced the intelligibility of one recording condition more than another. An analysis of the variance, summarized in table 1, indicated that there was no such differential effect and that, consequently, the intelligibility associated with any one of the three recording conditions might be represented by a single mean.[3] The mean intelligibility for coryzal speech, represented by the first encircled dot in the figure, was 62.02 (standard deviation, 9.93); for treated-coryzal speech (second dot), 60.83 (standard deviation, 8.19) and for normal speech (third dot), 64.42 (standard deviation, 7.20). Analysis by the method of Fisher, summarized in table 2, indicated that these amounts were probably not random variations of

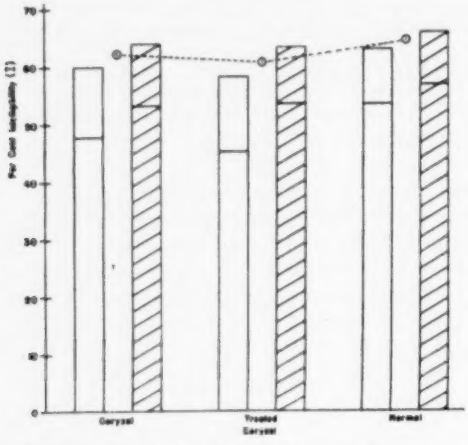

Chart 1.—Means and standard deviations of six distributions, each of which consisted of intelligibility scores of 24 subjects for one of three recording conditions and one of two playback conditions. The open bar indicates use of a loudspeaker in a quite room; the striped bar, use of headphones in a noise-filled room; the dot, mean of two playback conditions; the top of the bar, mean of the intelligibility scores; the horizontal line within the bar, the mean minus 1 standard deviation.

a single population value. Although it did not appear improbable that intelligibility of coryzal speech and treated coryzal speech are the same, it would seem that there might be a difference between the latter of these and the intelligibility of normal speech. It should be noted, however, that the obtained difference was not highly significant.[4]

3. $V_{cp} < V_{cpn}$.

4. Since the second recording of each subject was made within several minutes of the first, it is possible that there was an adaptation effect which diminished the variation among the means.

Examination of the intelligibility indexes of each subject separately revealed information not made obvious by consideration of only the group averages. Although the average intelligibility obtained for the coryzal speech condition was slightly higher than that obtained for the treated-coryzal recordings, 13 speakers had better intelligibility after receiving the nasal spray than before and 11 were less intelligible. And, although the mean for the coryzal speech was lower than that for the normal, 14 speakers were more intelligible when they had a cold than when they were well, whereas only 10 were less intelligible.

TABLE 1.—*Analysis of Variance for Speech Intelligibility Scores*

Source of Variation	Degrees of Freedom	Variance Estimate
Recordings (r)	2	159.92
Playbacks (p)	1	556.17
Subjects (s)	23	232.14
Interaction rp	2	11.11
Interaction rs	46	38.42
Interaction ps	23	286.19
Interaction rps	46	78.87
	143	

TABLE 2.—*F and t Evaluations of Differences Between Means Based on Combined Intelligibility Scores (Loudspeaker-Quiet plus Headphone-Noise) for Three Recordings: (1) Coryzal, (2) Treated Coryzal and (3) Normal (N-24)*

$V_r/V_{rs} = 159.92/38.42 = 4.163$ ($P < 0.05$)
$(M_3 - M_1)/\text{S.E.}_{\text{diff.}} = 2.40/2.218 = 1.080$ ($P < 0.30$)
$(M_3 - M_2)/\text{S.E.}_{\text{diff.}} = 3.59/1.683 = 2.129$ ($P < 0.05$)
$(M_1 - M_2)/\text{S.E.}_{\text{diff.}} = 1.19/1.946 = 0.674$ ($P < 0.60$)

In an attempt to find an explanation for these discrepancies, the direction of change shown by each speaker in his intelligibility was compared with the amount and type of each of four medical symptoms. There seemed to be little relation between direction of shift in intelligibility from that of coryzal speech to that of either treated-coryzal or normal and extent of nasal inflammation, nasal edema or nasopharyngeal inflammation (table 3). Possibly it is noteworthy that, although marked nasal discharge was observed in nearly half of the subjects whose shift in intelligibility from the coryzal to the treated-coryzal condition followed the direction of the averages, only 3 of the subjects whose shifts were in the opposite direction displayed the same effect, whereas 10 revealed little or no nasal discharge. Similarly, although considerable nasal dis-

charge characterized half of the subjects whose coryzal intelligibility was —as in the case of the means—lower than their normal intelligibility, of those whose differences between coryzal and normal intelligibility departed from the pattern of the means, only 3 presented marked nasal discharge while 11 presented relatively little. Neither of these discrepancies, however, was great enough to produce a chi-square value which accompanies the commonly accepted level of significance.[5]

There are at large a number of persons who on meeting some one displaying obvious coryzal symptoms seem unable to resist remarking in an unnatural voice, "Goodess be! You got a cold id your doze." The implication of such a cliche—that sounds requiring nasal emission

TABLE 3.—*Distribution of Speakers According to Direction of Change in Intelligibility and According to Extent of Each of Four Coryzal Symptoms During Cold*

	Nasal Inflammation *		Nasal Edema *		Nasal Discharge *		Pharyngeal Inflammation *	
	+	0	+	0	+	0	+	0
A. Coryzal vs. Treated Coryzal								
$Ic > Ix$	4	7	6	5	5	6	6	5
$Ic < Ix$	5	8	7	6	3	10	7	6
B. Coryzal vs. Normal								
$Ic > In$	6	8	8	6	3	11	7	7
$Ic < In$	3	7	5	5	5	5	6	4

* A marked condition is indicated by + and a slight or nonexistent condition by 0.

are altered by a cold—suggested the possibility that any influence of coryza on intelligibility may be greater for words which include nasal sounds than for other words. In order to evaluate this possibility, each of the three lists recorded by each of the 24 subjects was considered as consisting of two sublists: one made up of words containing one or more of the three nasal phonemes, the other made up of words containing only phonemes ordinarily uttered with velopharyngeal closure.[6] For each subject, the listener identifications for the two playback conditions were combined, and two subscores—one for each sublist—were computed for each of the three recording conditions.

Means and the standard deviations for the six distributions of subscores are represented in chart 2; the distributions of over-all sub-

5. Chi-square values were 1.34 ($P = 0.25$) for the first discrepancy mentioned and 2.16 ($P = 0.15$) for the second.

6. Nasal-including words—i. e., those containing *m*, *n* or *ng*—constituted about 35 per cent of the total in most of the lists used.

scores, each of which was a subject's average for all three recording conditions, are similarly represented for the two word-types. Under all three conditions of speaking, the average subject was more intelligible when he read nasal-including words than when he read other words; the difference was considerably greater, however, when he did not have a cold. This variation in amount of difference, judging from the statistical analysis which is summarized in table 4, might not be representative of the populations sampled.[7] Indeed, all such differences may be negligible.[8]

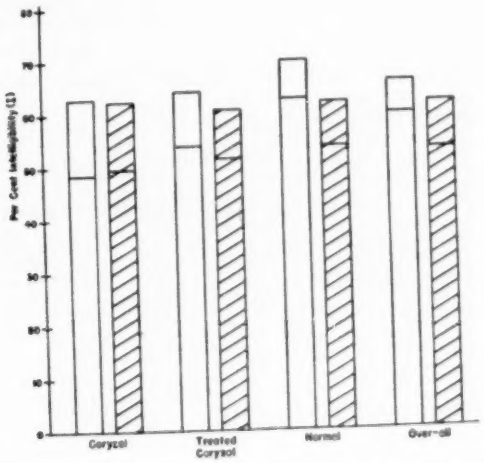

Chart 2.—Means and standard deviations of six distributions, each of which consisted of intelligibility subscores of 24 subjects for one of three recording conditions and one of two word types, and of two distributions, each of which consisted of over-all subscores of 24 subjects for one word type. The open bar indicates nasal-including words; the striped bar, words containing no nasal sounds; the top of the bar, the mean of the intelligibility subscores; the horizontal line within the bar, the mean minus 1 standard deviation.

Although it was not the purpose of the investigation to evaluate the influence of phonetic content on speech intelligibility, it may be of interest to point out that the over-all average intelligibility of the nasal-including words (65.24) was significantly greater than that of the other words (61.24).[9] This difference is made all the more interesting by the fact

7. $V_{rw}/V_{rws} = 1.77$ $(P > 0.05)$.
8. $V_r/V_{rs} = 2.19$ $(P > 0.05)$.
9. $V_w/V_{ws} = 8.92$ $(P < 0.01)$.

that it contradicts findings of Mason, who reported means for words containing the sounds [*m*] and [*n*] that were significantly smaller than means for other words.[10] Possibly pertinent here is the fact that Mason's intelligibility indexes were based on utterances through low fidelity carbon microphones, which are generally held so close to the lips that there is little chance for nasal sounds to be transmitted.

Recognizability.—Another aspect of speech, other than intelligibility, for which listeners furnished data was recognizability of quality. From the original recordings two dubs were made, each containing a pair of three word samples for each of the 24 subjects. The first dub consisted of words from the "coryzal" recordings paired against words from the "normal" recordings in random order. The second dub consisted of words from the "coryzal" records paired against words from the

TABLE 4.—*Analysis of Variance for Speech Intelligibility Subscores Represented in Chart 2*

Source of Variation	Degrees of Freedom	Variance Estimate
Recordings (*r*)	2	218.75
Word types (*w*)	1	580.00
Subjects (*s*)	23	213.67
Interaction *rw*	2	172.30
Interaction *rs*	46	99.84
Interaction *ws*	23	66.08
Interaction *rws*	46	97.82
	143	

"treated-coryzal" records. Both dubs were played to 72 listeners who had been instructed to identify, guessing if necessary, the coryzal utterance of each pair. The results are summarized in chart 3.

The coryzal utterances of most of the speakers were distinctive enough, compared with their normal speech, to be correctly identified by a majority of the listeners. The coryzal words of 5 of the subjects, however, sounded more normal to most of the listeners than did the samples furnished by those subjects after their colds had disappeared. Indeed, in the case of 1 speaker, four fifths of the listeners labeled the normal utterance as coryzal. On the other hand, over 90 per cent of

10. Mason, H. M.: Phonetic Characteristics of Words as Related to Their Intelligibility in Aircraft Noise, report no. 4681, PB no. 12160, Office of Scientific Research and Development, United States Emergency Management Office, Feb. 10, 1945. The data of this study along with findings of another study are also reported in a publication by Stevens, W. E.: Study of Phonetic Factors in Relation to Accuracy of Transmission of Words in Airplane Noise, Speech Monogr. **13**:34-40, 1946.

the listeners were right in their judgments concerning the speech samples of 2 of the speakers. The average pair of utterances was correctly judged by about two thirds of the listeners.

Evidently identifying speech samples uttered by persons with colds before and after vasoconstriction is a more difficult task than distinguishing between coryzal and normal speech. When presented along with words representing the treated-coryzal condition, the coryzal utterances of only 11 subjects were recognized as such by 50 per cent or more of the judges. The coryzal words of the other 13 speakers were mistaken for treated-coryzal utterances by a majority of the listeners. In only 2 cases was coryzal speech correctly identified by

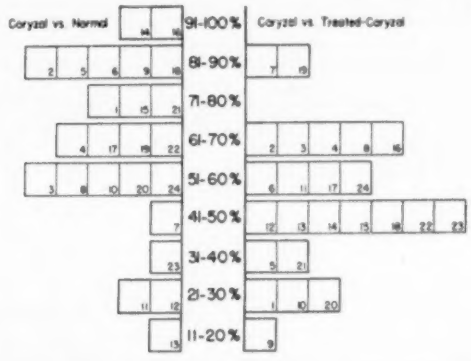

Chart 3.—Distribution of 24 speakers according to percentage of 72 listeners distinguishing coryzal speech from (1) normal speech and from (2) treated-coryzal speech. Each square represents 1 speaker, and individual speakers are identified by numbers within the squares.

more than 70 per cent of the listeners. The average pair was judged correctly by the same proportion of listeners as judged it incorrectly.

Although the coryzal speech of a speaker might be recognized by a large number of listeners comparing it with normal speech, it might be recognized by but a few listeners comparing it with treated-coryzal speech. Subject 1, for example, had 76 per cent of the listeners correctly identify his coryzal speech when it was contrasted with his normal speech; whereas, only 30 per cent correctly distinguished it from his treated-coryzal speech. The correlation coefficient obtained in an effort to describe the relationship between the number of listeners who responded correctly in the "coryzal *vs.* normal" comparison and the

number who responded correctly in the "coryzal *vs.* treated-coryzal" comparison was insignificantly larger than zero ($r = 0.048$).[11]

In the hope that the variation among the 24 subjects with respect to recognizability of coryzal speech might be better understood, the data previously presented were compared with the medical findings. Results are summarized in table 5. Of particular concern were those subjects whose coryzal utterances were mistaken for their normal or treated-coryzal speech samples by more than 50 per cent of the listeners. Of the 13 whose coryzal and treated-coryzal utterances were confused by a majority of the listeners, marked nasal discharge had been observed in only 3. Chi-square tests, however, indicated that any apparent associ-

TABLE 5.—*Distribution of Speakers According to Relative Incidence of Right and Wrong Quality Recognitions and According to Extent of Four Coryzal Symptoms During Cold*

	Nasal Inflammation *		Nasal Edema *		Nasal Discharge *		Pharyngeal Inflammation *	
	+	0	+	0	+	0	+	0
A. Coryzal vs. Treated-Coryzal								
$R > W$	3	8	5	6	5	6	8	3
$R < W$	6	7	8	5	3	10	5	8
B. Coryzal vs. Normal								
$R > W$	8	11	10	9	6	13	9	10
$R < W$	1	4	3	2	2	3	4	1

* A pronounced condition is indicated by +; a slight or nonexistent condition by 0.

ation between this—or between severity of any of the three intranasal symptoms—and proportion of correct identifications of coryzal speech might easily be explained as a function of sampling error. Possibly more significant is the fact that 8 of these 13 speakers were found to have relatively little nasopharyngeal inflammation, whereas all but 3 of the remaining 11 subjects were found to have a considerable amount.[12] Interestingly enough, of the 5 speakers whose coryzal words were distinguished from their normal utterances by only a minority of the listeners, marked nasopharyngeal inflammation was noted in all but one; however, this condition was observed in slightly less than half of those whose coryzal speech was evidently more distinctive.[13]

11. Of possible interest here is the fact that the number of paired speech samples correctly judged by a listener in one comparison was not a good indication of the number he might judge right in the other comparison ($r = -0.061$).

12. Chi-square = 2.82 ($P = 0.09$).

13. Chi-square = 1.69 ($P = 0.19$).

Although no attempt was made to determine all the phonetic components of the utterances used in the two quality comparisons, the incidence of the normally nasal sounds was noted for each of the four samples which represented each subject. These data combined with the determination of which subjects had coryzal speech mistaken for either treated-coryzal or normal speech by a majority of the listeners made possible the compilation of table 6. In twelve of "coryzal *vs.* treated" pairs, the coryzal contained more nasal sounds than did the treated-coryzal utterance; in the other twelve pairs the latter contained an equal or greater number of nasal sounds than did the coryzal. Both groups included about the same number of pairs which were misidentified by 50 per cent or more of the listeners. In the other comparison, the coryzal member of thirteen pairs of utterances contained more nasal sounds than did the normal sample; in the remaining eleven pairs the

TABLE 6.—*Distribution of Speakers According to Relative Incidence of Right and Wrong Identifications of Coryzal Speech and According to Relative Occurrence of Nasal Phonemes in the Speech Samples*

	A. Coryzal vs. Treated-Coryzal		B. Coryzal vs. Normal	
	$Nc > N_T$	$Nc \leqq N_T$	$Nc > N_N$	$Nc \leqq N_N$
$R > W$	5	6	11	8
$R < W$	7	6	2	3

latter contained an equal or greater number of such sounds compared with the coryzal. The five pairs which were misidentified by a majority of the listeners were as evenly divided between the two groups as their odd number would allow. These distributions obviously furnish no basis for associating coryzal speech which is indistinguishable from other kinds of speech with the relative number of nasal sounds it may contain.

As was stated earlier, the coryzal sample of all but 5 speakers was distinguished from its normally spoken counterpart by at least half of 72 listeners. In the case of any pair of utterances it is possible, in view of the listeners' knowledge of the two conditions which the pair represented, that many of the correct judgments resulted from a decision on the part of many listeners that the utterance which sounded the less "natural" must be coryzal speech even though it might not sound similar to other utterances judged as coryzal on the same basis. In other words, it is possible that the coryzal utterances had nothing more in common than abnormality and that the abnormal aspect of each such utterance might have been a unique characteristic. On the other hand, it is possible that those utterances which were classified as coryzal by most of the judges all had in common one or more acoustic properties. The latter possibility led to the carrying out of another experiment.

Four distorted dubs were made of the "coryzal *vs.* normal" recording, which could reproduce all frequencies between 50 and 8000 cycles with good fidelity. Each dub was made by recording the output of an adjustable low pass filter set [14] to which was fed the output of a phonograph playing the high fidelity version. For one dub the filter set was adjusted to attenuate sharply all frequencies above 4400 cycles; for another, all frequencies above 3000 cycles; for another, all above 2200 cycles, and for the fourth, all above 1000 cycles. Thus, counting the one from which the others were made, there became available five recorded versions of

TABLE 7.—*Distribution of Subjects According to Level of Significance Accompanying Chi-Square Tests of the Shift Found for Each in the Number of His Listeners Who Correctly Differentiated Between His Coryzal and His Normal Speech When the Basis of Differentiation Shifted from Relatively Full Range Phonograph Records to Records Sharply Attentuated in All Harmonics Above 4400, 3000, 2200, or 1000 Cycles per Second (N = 24).*

	Level of Significance									
Cycles	0.10	0.20	0.30	0.40	0.50	0.60	0.70	0.80	0.90	1.00
4400	0	5	2	0	2	1	3	6	1	4
3000	0	1	2	1	2	6	3	4	0	3
2200	2	0	5	2	1	0	4	5	0	5
1000	1	3	2	1	3	6	1	2	1	4

coryzal utterances paired against normal utterances, the versions all alike except for width of frequency range. Forty midshipmen, none of whom were among the listeners previously used, were called on to listen to the five records and to identify the coryzal samples, as had the seventy-two listeners previously mentioned. The forty were divided into panels of eight. One panel heard one record; then another panel heard a different record, and so on until finally—at the end of twenty-five listening sessions—all five panels had responded to all five records but in different sequence. At least thirty minutes separated any two sets of responses by a single panel.

The number of recognitions of coryzal speech was determined for each recorded representation of each speaker. From these data four 2×2 contingency tables were prepared for each speaker, each table showing the number of correct and incorrect responses to both the full-range and one of the distorted representations of that speaker. The discrepancy in each table between the ratio of right to wrong responses accompanying one record and the comparable ratio accompanying the other record was evaluated by means of a chi-square test. The results

14. Type RA — 243, Electrical Research Products, Inc.

of the 72 evaluations thus made are summarized in table 7. A glance at the table will reveal that cutting off the upper frequencies—whether at 4400 or at 1000 cycles or at a point in between—of both a coryzal and a normal utterance is not likely to increase the difficulty of identifying the samples. In the case of only 1 speaker could it be said with confidence at the 5 per cent level that listeners responded differently to two records of different frequency ranges.

These data do not imply that the coryzal utterances do not have some common acoustic characteristic by which they could be differentiated from normal speech. It is possible that differences might have been obtained had records been dubbed which had altered the spectrums of samples below 1000 cycles. This possibility seems all the more tenable when one considers the results obtained by Kelly when he analyzed the energy distribution of vowels uttered by a speaker whose nasal resonance had been eliminated by nasopharyngeal packing.[15]

Relation Between Intelligibility and Recognizability.—In the preceding sections of this paper it has been reported that a large proportion of the subjects on catching a cold acquired a different sort of vocal quality and almost as many of them became less intelligible. In view of these findings it seemed reasonable to hypothesize that the greater the proportion of listeners who could distinguish between a sample of a man's coryzal speech and one of his normal speech, the more sizable would be the difference between his coryzal and his normal intelligibility score. In order to test this hypothesis, a difference based on the subtraction of the coryzal intelligibility score from the normal score was determined for each subject, and the differences thus obtained were correlated with the corresponding proportions of correct recognitions obtained from the "coryzal *vs.* normal" quality comparison. The correlation coefficient thus obtained was — 0.34, an amount somewhat less than what is generally regarded as significant.[16]

A hypothesis similar to that stated in the preceding paragraph but pertaining to the difference in intelligibility between coryzal and treated-coryzal speech and to recognizability of coryzal speech when contrasted with treated-coryzal speech was also evaluated. The resulting correlation coefficient was 0.10, a negligible value.

Although neither of the correlation coefficients was of significant magnitude, their signs give them some interest. It will be recalled that the mean coryzal intelligibility score was somewhat smaller than the mean normal score; it would seem reasonable to assume, therefore, that

15. Kelly, J. P.: Studies in Nasality, Arch. Speech **1**:34-35, 1934.

16. Values of r required for significance at the 1 per cent and 5 per cent levels are, respectively, 0.515 and 0.404.

any effect of a cold on intelligibility would be an adverse one. In view of this consideration and the fact that the differences which constituted one set of values in the first correlation previously mentioned were obtained by subtracting the coryzal score from the normal score, it would seem that the coefficient should have been a positive one rather than the negative one that was obtained. A similar line of reasoning would suggest that the other coefficient should have been negative rather than positive.

CONCLUSIONS

1. Whether their words are transmitted to listeners through earphones in a very noisy room or through a loudspeaker in a quieter place, a group of men with coryza will vary considerably with respect to direction and amount of change that administration of a vasoconstricting nasal spray will produce in the intelligibility of their speech.

2. Their average intelligibility, however, after such treatment will probably be lower than a comparable average representing their normal speech, provided that no more than about a third of their words contain sounds ordinarily requiring nasal emission.

3. Prominence of any symptom in the nose or nasopharynx of a man with a cold will tell little of what temporary vasoconstriction or complete recovery would do to the intelligibility of his speech.

4. Coryzal utterances of most men can be distinguished from their normal utterances by a majority of untrained listeners; however the coryzal speech of some men is rather misleading.

5. When presented with a number of pairs of speech samples, a majority of listeners—although aware that one of each pair was spoken by a man with a cold shortly after his nasal passages had been sprayed and that one had been spoken before such treatment—are likely to be wrong in at least half of their attempts to identify coryzal speech.

6. Although a man's coryzal utterance might be recognized by a large proportion of listeners comparing it with his normal speech, it might be recognized by relatively few listeners comparing it with something he said shortly after his nose had been sprayed.

7. Conspicuousness of any intranasal condition, such as inflammation, edema or discharge, is not an indicator of the proportion of listeners who will be able to distinguish a man's coryzal speech from utterances he might make after he is given a vasoconstricting nasal spray or from those he might make after his cold disappears.

8. There may be some relationship between distinctiveness of coryzal vocal quality and extent of nasopharyngeal inflammation.

9. There is no relationship between the incidence of nasally emitted phonemes in coryzal speech and its distinguishability from the other kinds of speech previously mentioned.

10. Attenuation of harmonics above 1000 cycles in a sample of coryzal speech is not likely to alter the proportion of listeners who could distinguish it from a comparable sample of normal speech.

11. There may be little or no relation between the amount of influence catching a cold might have on a man's normal intelligibility and the distinctiveness of his coryzal speech.

Speech and Hearing Clinic, University of Southern California.

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EDGAR I. STEINBERG, M.D.
PHILADELPHIA

REPORTS [1] from the Academy of Sciences of the Ukrainian Socialist Soviet Republic in 1942 on the effective therapeutic use of antireticular cytotoxic serum have presented it as a new agent for clinical use in many pathologic processes and, in particular, for such otolaryngologic conditions as atrophic rhinitis. Antireticular cytotoxic serum for use in man is produced by intravenous injection into animals of a saline extract of spleen and bone marrow taken after death from persons who had died from injury and who had had no acute or chronic disease. The tissues are obtained within ten hours after death from persons not more than 40 years old. The antiserum developed in animals is used in treating diseases in man in which it seems necessary to stimulate the trophic, plastic or protective functions of the physiologic system of the reticuloendothelial tissue. Mihailovsky, Smirnova and Marchuk [2] reported the effects of antireticular cytotoxic serum used in 18 cases of ozena and 23 cases of rhinopharyngitis. Kravchenko reported the results in 120 patients with rhinopharyngitis and 86 with ozena. The effect manifested itself by changes in the character of the discharges, decrease in their amount and disappearance of bad odor; in all cases improvement was noted within four to six weeks after completion of treatment. The only reaction noted was local cutaneous hyperemia in a few cases, but this disappeared within twenty-four to forty-eight hours.

From the Department of Otolaryngology, Graduate Hospital of the University of Pennsylvania, and Children's Hospital of Philadelphia.

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1. Abstracts of reports delivered at the Conference of the Academy of Sciences of the Ukrainian S. S. R., Ufa, July 1942.

2. Mihailovsky, S. V.; Smirnova, V. A., and Marchuk, P. D.: Treatment of Ozena with the Cytotoxic Antireticular Serum, read before the Academy of Sciences of the Ukrainian S. S. R., July 1942.

3. Footnote deleted.

In this country, Jackson [4] reported on his attempt to duplicate the work described by Kravchenko. In a group of 24 patients thus treated, the results fell far short of those reported by the Russians. Jackson made the following statement:

> It seems unlikely that atrophic rhinitis (ozena) is any different there [in Russia] than here, except that they may have reported only cases in their military age group. The preparation of the serum was identical in every detail to that of Bogomolets. The dosage recommended was exceedingly small (of the order recommended by Bogomolets) for a stimulating effect. . . . In some of the patients with ozena . . . there was some improvement; but there was no improvement in the larger percentage of cases. . . . Cases showing improvement were judged mostly on subjective phenomena. . . . No change was noted in the size of atrophic structures. . . . The cause of the difference between the results reported by the Russians and our results is not obvious . . . further observations on more cases is indicated for better evalution of the effect of antireticular cytotoxic serum in ozena.

Antireticular cytotoxic serum having been described both as efficacious and ineffectual in different conditions, both acute and chronic, as a review shows,[5] the present study was planned to test the effectiveness of this serum in the treatment of various otolaryngologic conditions of childhood.

Earlier general investigative therapy and antecedent work was first reported by Bogomolets,[6] an abstract of whose concepts and works may be briefly presented as follows:

> The concept of the reticuloendothelial tissue of the body as a physiologic system, though anticipated by numerous scientists (beginning with the brilliant investigations of Ranvier; the macrophagic system of Mechnikov, the reticuloendothelial system of Aschoff, etc.), will require some explanation. The term "physiologic system of the reticuloendothelial tissue" was proposed in 1924 in Bogomolets' work "The Constitution and the Mesenchyme." The histologic elements of the reticuloendothelial tissue include clasmatocytes, reticular cells, fibroblasts, adventitial cells, basophilic cells, oxyphilous plasma cells, fatty

4. Jackson, C. C. R.: Antireticular Cytotoxic Serum in Atrophic Rhinitis (Ozena), Laryngoscope **56**:342-351 (July) 1946.

5. Bogomolets, A. A., and Marchuk, P. D.: Antireticular Cytotoxic Serum in Therapy of Scarlet Fever: Preliminary Report, Vrach. delo **19**:831-834, 1936; Med. zhur. **6**:689-692, 1936. Straus, R.: Antireticular Cytotoxic Serum; Introduction and Review of Literature, J. Immunol. **54**:151-154 (Oct.) 1946.

6. Bogomolets, A. A.: Antireticular Cytotoxic Serum as a Means of Pathogenetic Therapy, Am. Rev. Soviet. Med. **1**:101-112 (Dec.) 1943; Stimulating Effect of Antireticular Cytotoxic Serum on Physiologic System of Connective Tissue, Med. zhur. **9**:789-792, 1939; Antireticular Cytotoxic Serum: Therapeutic Activity, Soviet med. (no. 1) **7**:1-4, 1943; Antireticular Cytotoxic Serum, Brit. M. J. **2**:203 (Aug. 14) 1943; Antireticular Cytotoxic Serum as a Means of Pathogenic Therapy, Canad. M. A. J. **52**:76-77 (Jan.) 1945.

cells rich in lipase, chromatophores, epithelioid and granulomatous giant cells, reticuloendothelial elements of the spleen, monocytes, lymphocytes and osteoblasts.

Reticuloendothelial tissue possesses the following functions: 1. Trophic regulation of cellular nutrition and metabolism, formation maintenance of the hematoparenchymal barrier and the function of a depot. Schade drew attention to the significance of intercellular substances and to their role as semipermeable membranes through which metabolism takes place between cells and body fluids. These intercellular substances are produced by reticuloendothelial tissue. 2. Plastic participation in the regeneration of tissues and in the healing of wounds, ulcers and fractures. 3. Protective (reaction to infection by phagocytic and organizing activity at the site where antibodies are produced and participation in the reaction toward new growths). 4. Autoregulative (formation of hormones, especially in the spleen). 5. Mechanical (osseous and elastic skeleton formed in part by the reticuloendothelial tissue).

Since the reticuloendothelial tissue is the arena in which pathologic processes develop, the maintenance of this system is one of the most important problems in therapy. In 1900 Mechnikov and Bezredka increased the formation of erythrocytes by injecting small doses of hemolytic serum and caused leukocytosis by injecting leukotoxic serum. Mechnikov's chief obstacle was his inability to determine the titer of cytotoxins in serum. In 1908 Bogomolets had the same difficulty while using cytotoxic adrenal serum, but after several years it was found that the titer of cytotoxins could be determined with the complement fixation test, using the same antigen for both fixation of the complement and immunization. Thus he prepared "antireticular serum" by injecting human spleen and bone marrow into animals and subsequently obtaining the animals' serum.

Exceedingly small doses of the serum are used in man, since larger doses inhibit the physiologic system. After injection of serum there is increased permeability of the hematoparenchymal barrier, dilatation of capillaries and relative lymphocytosis. The lymphocytosis disappears after three or four hours and is replaced by monocytosis. The duration of this condition also is brief, although monocytes are sent in increased numbers to tissues which require them for several days. Some effects observed with the use of antireticular cytotoxic serum are as follows: 1. Blood pressure in hypertensive cases is permanently lowered. The treatment must be applied before irreversible sclerotic changes have occurred in the hematoparenchymal barrier. 2. The healing rate of fractures is greatly accelerated. Large doses of the serum delays healing. 3. It reduces the relapse rate after operation for cancer of the stomach and bronchus. (Professor Kavetski and associates showed an increased carcinolytic serum index after serum injection. This index is lower

than normal in patients with cancer.) 4. It increases the complement of the blood. 5. It increases the opsonic index of the blood. 6. The erythrocyte sedimentation rate is restored to normal in many cases by its altering of the hematoparenchymal barrier so that proteins formed by the physiologic system of the connective tissue can enter the blood stream. 7. It prevents scarlet fever when given early and modifies the course of the disease when given later.

Bogomolets hoped that the serum would not only mobilize the plastic and protective energy of the reticuloendothelial tissue, but would also prolong its trophic function, thus preventing precocious senile fading of the body. (He believes that normal longevity is 125 to 150 years.)

In a conference on antireticular cytotoxic serum held in Ufa on July 12, 1942, a resolution was passed stating that this serum was a powerful specific factor influencing the reticuloendothelial tissue. On the basis of more than 2,500 clinical observations presented, indications for use of this serum were clearly established for many conditions, such as tonsillitis, and diseases connected with disordered trophic function of tissue, such as ozena. The members of the conference stated that it was absolutely necessary that antireticular cytotoxic serum be used extensively in hospitals.

This and other reports which have appeared in the past have been concerned with various clinical entities. However, it was obvious that in pediatric otolaryngology one deals with many cases of dysfunction of portions of the reticuloendothelial system. I decided, therefore, to investigate the use of antireticular cytotoxic serum as therapy in pediatric otolaryngology. This was not undertaken without some mental reservations, for in a personal communication from Culbertson,[7] I learned that he had attempted to repeat some of the work of the Russian authors and, on a series of "exploratory" studies, he had not found enough encouragement to go on with more extensive work than had been done. He felt that the results obtained in this country were not promising and that experiments with antireticular cytotoxic serum compared to controlled experiments with normal serums had been rather disappointing. Parenthetically, he stated, "It is, of course, to be remembered that we may not have prepared the serum in a fashion which was exactly correct, but we attempted to follow the Russian directions in detail. We know that we had serum which gave potent complement fixation tests. We have not been able to produce demonstrable changes in animal tissues nor, in the few cases we have studied, notable effects in human beings."

Antireticular cytotoxic serum prepared in the following manner (according to the method of Marchuk [8]) was used in the present study.

7. Culbertson, C. G.: Personal communication to the author.

8. Marchuk, P. D.: Method of Preparing and Preserving Antireticular Cytotoxic Serum, Am. Rev. Soviet Med. **1**:113-123 (Dec.) 1943.

Antigen is obtained from the marrow of the spleen and ribs of a human cadaver not later than six to ten hours after death. Five to 15 Gm. of tissue, in the proportion of one to two parts of bone marrow to eight or nine parts of spleen, are minced with a knife and washed with isotonic sodium chloride solution to extract the erythrocytes. The tissue is then ground thoroughly in a porcelain mortar and five times its weight of isotonic sodium chloride solution added. Half of the resulting suspension is centrifuged for four minutes at 1,000 revolutions per minute. The supernatant fluid, from which the upper layer of fat has been removed, is injected intravenously into a rabbit, and the uncentrifuged antigen is introduced orally. The antigen is injected five or six times in increasing doses at intervals of from four to five days, and eight or nine days after the last injection, the titer of cytotoxins in the rabbit's serum is determined by complement fixation. The serum is suitable for use if its titer is not less than 1 : 100 and the hemolytic titer in relation to human erythrocytes is not more than 1 : 16. Blood is then drawn from the rabbit, and the serum is separated and stored in ampuls. It is cultured to ascertain its sterility, and its toxicity is checked on guinea pigs by intravenous and subcutaneous injection in amounts of 10 cc. It is diluted 1 : 10 with sterile saline solution before injection, and the human dose is 0.1 to 0.25 cc. The diluted serum must be kept in the dark at 4 C. and used within ten days of dilution.

A series of injections consists of three injections of the dilute serum. The injections are given subcutaneously, usually in the deltoid region. A new series of injections is given in six weeks, except in special cases. Local reactions (erythema, edema, pruritus) may develop, and there may be a general reaction of fever and even nausea. These reactions are uncommon, however, and only conservative symptomatic treatment need be used. In some sensitive patients who have shown a severe reaction to one or more injections of the first series, the reaction has been obviated or reduced in a subsequent series by the administration of diphenhydramine hydrochloride (benadryl hydrochloride®) prior to the injections. The use of antireticular cytotoxic serum is contraindicated with the following conditions: acute endocarditis and myocarditis, nephrosis and nephritis, bronchial asthma, and exudative forms of tuberculosis.

Clinical observations on 25 cases and 2 controls were made in the outpatient department, service of Dr. Karl M. Houser, at Children's Hospital, Philadelphia, and Graduate Hospital of the University of Pennsylvania. At intervals of two or three days, antireticular cytotoxic serum was administered. The first series of injections consisted of doses of from 0.25 to 0.5 to 0.75 cc. After a period of four to six weeks, many patients received a second series of from 0.5 to 1.0 to 1.0 cc.

Slight changes, such as omission of other treatment previously administered, which often consisted of local therapy, were made during the period of observation. To avoid undue reliance on reports of subjective improvement, objective physical examination and laboratory studies were recorded before, during and after therapy with antireticular cytotoxic serum (see table).

COMMENT

Mild reactions of a transitory character occurred at the site of injection in 2 boys and in 2 girls in this study. These local reactions consisted of erythema, edema and slight pruritus, which required nothing more than symptomatic therapy. In this group some amelioration of the symptoms followed these reactions in 1 boy and 1 girl.

The effects observed from the treatment in this series of 17 boys and 10 girls with various otolaryngologic conditions were, on an over-all evaluation, rather slight and inconclusive. Further investigation does not seem to be definitely justified. Despite the small number of patients treated, the preponderance of negative results obtained in this report is in complete agreement with other recent observations on the use of this serum in other fields. This paper is presented to place these results on record as part of the observed experiences with this form of treatment.

SUMMARY AND CONCLUSION

This paper presents a review of the literature and a study of clinical observations on pediatric patients in the nose, throat and ear service of the outpatient department of two hospitals in Philadelphia.

Observations on 27 pediatric patients, 17 boys and 10 girls, were performed over a period of one year. Two children presented no definite problem and served as controls; 11 of those with various otolaryngologic conditions received one series of injections, and 16 patients received two series of injections.

Ten patients who received only one series of injections experienced no objective or subjective change in their symptomatology. One child who received only one series derived the greatest benefit observed from this form of therapy.

Eleven patients who received two series of injections experienced no subjective or objective change. Four patients thought they had improved, but no changes were seen on physical examination. One patient showed slight improvement and one moderate improvement, both subjectively and objectively.

From the results obtained here and from a review of the literature it may be concluded that there does not seem to be a definite indication for antireticular cytotoxic serum in pediatric otolaryngology.

Summary of Data on 27 Patients Treated with Injections of Antireticular Cytotoxic Serum

Case	Birth Date	Sex	Diagnosis	Length of Illness	Number of Series Given	Dates of First Series of Injections	Dates of Last Series of Injections	Date of Last Observation	Reaction to Serum*		Laboratory Data†
									Subjective Report	Objective Observations	
1	8/ 4/42	M	Chronic tonsillitis and adenoiditis	2 yr.	1	11/24/48 11/26/48 11/29/48	....	5/25/49	U	U	Blood: Hb. 12.5 Gm.; RBC, 4,440,000; WBC, 12,300; N. 94%, L. 25%, M. 1%; serologic reactions negative
2	11/11/39	F	Fever of unknown origin and hypertrophy of tonsils and adenoids	19 mo.	2	11/24/48 11/26/48 11/29/48	2/ 9/49 2/16/49 2/19/49	4/30/49	U	U	Blood: Hb. 11 Gm.; RBC, 4,380,000; WBC, 4,300; N. 42%, L. 49%, M. 4%, E. 5%; sedimentation rate 30 mm. per 60 m.n.
3	5/30/43	F	Chronic rhinosinusitis	6 mo.	2	11/29/48 12/ 1/48 12/ 3/48	2/ 7/49 2/14/49 2/21/49	4/13/49	U	U	Blood: Hb. 10 Gm.; RBC, 3,770,000; WBC, 6,350; N. 28%, L. 6%, M. 1%, E. 5%; serologic reactions negative
4	5/ 7/47	M	Well	13 mo.	1	12/ 1/48 12/ 3/48 12/ 8/48	....	12/27/48	U	U	Blood: Hb. 12 Gm.; RBC, 4,400,000; WBC, 9,100; N. 40%, L. 59%, M. 1%, E. 1%; nasal smear negative
5	12/25/46	M	Lymphoid hyperplasia	3 wk.	2	12/ 3/48 12/ 6/48 12/ 8/48	1/31/49 2/ 2/49 2/ 9/49	4/13/49	U	U	Blood: Hb. 9 Gm.; RBC, 4,400,000; WBC, 7,600; N. 46%, L. 50%, M. 3%, E. 1%; serologic reactions negative
6	1/19/37	M	Chronic suppurative otitis media	10 yr.	2	12/ 3/48 12/ 6/48 12/ 8/48	1/31/49 2/ 2/49 2/ 9/49	5/11/49	S	S	Blood: Hb. 11 Gm.; RBC, 3,700,000; WBC, 5,300; N. 57%, L. 39%, M. 1%, E. 3%; serologic reactions negative
7	10/24/42	M	Chronic rhinopharyngitis	8 wk.	2	12/ 3/48 12/ 6/48 12/ 8/48	1/31/49 2/ 2/49 2/ 9/49	3/23/49	S	U	Blood: Hb. 10 Gm.; RBC, 3,200,000; WBC, 8,100; N. 52%, L. 41%, M. 6%, E. 1%; serologic reactions negative
8	6/20/47	M	Recurrent nasopharyngitis	19 mo.	2	12/ 6/48 12/ 8/48 12/10/48	1/26/49 2/ 2/49 2/ 9/49	5/13/49	U	U	Blood: Hb. 13 Gm.; RBC, 4,300,000; WBC, 5,600; N. 49%, L. 49%, M. 1%, E. 1%; serologic reactions negative
9	2/14/46	F	Chronic rhinosinusitis	11 mo.	2	12/ 6/48 12/ 8/48 12/10/48	2/ 9/49 2/16/49 2/21/49	4/13/49	U	U	Blood: Hb. 11 Gm.; RBC, 3,500,000; WBC, 4,900; N. 37%, L. 58%, M. 5%, E. 1%; serologic reactions negative
10	2/25/37	F	Chronic suppurative otitis media	23 mo.	1	12/ 8/48 12/13/48 12/15/48	....	6/ 3/49	G	M	Blood: Hb. 12 Gm.; RBC, 3,900,000; WBC, 5,100; N. 63%, L. 28%, M. 5%, E. 3%; serologic reactions negative
11	5/ 2/43	F	Chronic tonsillitis	2 mo.	2	12/ 8/48 12/10/48 12/13/48	2/ 9/49 2/16/49 2/28/49	4/13/49	U	U	Blood: Hb. 11 Gm.; RBC, 3,400,000; WBC, 5,500; N. 40%, L. 50%, M. 4%, E. 6%; serologic reactions negative
12	4/22/36	M	Recurrent suppurative otitis media	15 mo.	2	12/10/48 12/13/48 12/15/48	2/ 7/49 2/21/49 2/28/49	4/13/49	M	M	Blood: Hb. 10 Gm.; RBC, 3,800,000; WBC, 10,500; N. 74%, L. 17%, M. 4%, E. 5%; serologic reactions negative
13	1/19/45	M	Allergic rhinosinusitis	9 mo.	2	12/13/48 12/15/48 12/17/48	2/ 2/49 2/ 9/49 2/16/49	5/13/49	S	U	Blood: Hb. 11 Gm.; RBC, 3,700,000; WBC, 5,300; N. 40%, L. 50%, M. 3%, E. 1%; serologic reactions negative
14	5/ 1/39	M	Hypertrophy of tonsils and adenoids	2 yr.	2	12/13/48 12/15/48 12/17/48	2/ 9/49 2/16/49 2/21/49	4/27/49	S	U	Blood: Hb. 11 Gm.; RBC, 4,700,000; WBC, 5,000; N. 50%, L. 45%, M. 4%, E. 1%; serologic reactions negative

Case	Birth Date	Sex	Diagnosis	Length of Illness	Number of Series Given	Dates of First Series of Injections	Dates of Last Series of Injections	Date of Last Observation	Reaction to Serum*		Laboratory Data†
									Subjective Report	Objective Observations	
15	8/ 1/39	F	Chronic lymphoid hyperplasia	8 yr.	2	12/13/48 12/20/48 12/27/48	2/ 9/49 2/16/49 2/21/49	5/ 6/49	U	U	Blood: Hb. 11 Gm.; RBC, 3,500,000; WBC, 5,100; N. 52%, L. 40%, M. 3%, E. 5%; serologic reactions negative
16	5/27/46	M	Chronic tonsillitis	Not known	1	12/17/48 12/20/48 12/27/48	....	5/20/49	U	U	Blood: Hb. 10 Gm.; RBC, 3,700,000; WBC, 4,500; N. 47%, L. 45%, M. 5%, E. 3%; serologic reactions negative
17	7/ 9/37	F	Chronic suppurative otitis media	2 yr.	2	12/20/48 12/22/48 12/27/48	2/ 2/49 2/ 9/49 2/16/49	4/23/49	U	U	Blood: Hb. 11 Gm.; RBC, 3,900,000; WBC, 8,200; N. 52%, L. 45%, M. 1%, E. 2%; serologic reactions negative
18	1/28/39	M	Hypertrophy of tonsils and adenoids	4 yr.	1	12/27/48 12/29/48 1/ 3/49	....	4/23/49	U	U	Blood: Hb. 12 Gm.; RBC, 4,300,000; WBC, 8,000; N. 60%, L. 35%, M. 1%, E. 4%; serologic reactions negative
19	9/ 7/43	M	Chronic perennial rhinosinusitis	2 yr.	2	12/27/48 12/29/48 1/ 3/49	2/ 9/49 2/16/49 2/23/49	4/23/49	U	U	Blood: Hb. 13 Gm.; RBC, 4,400,000; WBC, 10,500; N. 28%, L. 57%, M. 2%, E. 13%; serologic reactions negative
20	4/28/47	F	Chronic adenoiditis	13 mo.	1	12/27/48 12/29/48 1/ 3/49	....	4/23/49	U	U	Blood: Hb. 11 Gm.; RBC, 3,300,000; WBC, 7,100; N. 40%, L. 50%, M. 4%, E. 1%; serologic reactions negative
21	6/10/43	F	Chronic allergic rhinitis	1 yr.	1	12/27/48 12/29/48 1/ 3/49	....	4/29/49	U	U	Blood: Hb. 12 Gm.; RBC, 4,300,000; WBC, 4,700; N. 31%, L. 58%, M. 1%, E. 10%; sedimentation rate 23 mm. per 60 min.
22	4/ 4/46	M	Acute suppurative otitis media	5 wk.	1	12/27/48 12/29/48 1/ 5/49	....	2/23/49	U	U	Blood: Hb. 10 Gm.; RBC, 3,900,000; WBC, 7,200; N. 41%, L. 51%, M. 4%, E. 4%; sedimentation rate 27 mm. per 60 min.
23	10/23/36	F	Allergic rhinitis	8 yr.	2	12/27/48 1/ 3/49 1/ 5/49	2/ 2/49 2/ 9/49 2/16/49	4/23/49	S	U	Blood: Hb. 12 Gm.; RBC, 4,200,000; WBC, 5,400; N. 34%, L. 52%, M. 4%, E. 9%; serologic reactions negative
24	4/17/37	M	Well	7 mo.	1	12/27/48 12/29/48 1/ 3/49	....	2/23/49	U	U	Blood: Hb. 11 Gm.; RBC, 3,900,000; WBC, 5,000; N. 48%, L. 44%, M. 2%, E. 5%; serologic reactions negative
25	12/26/37	M	Chronic follicular tonsillitis	8 wk.	1	12/29/48 1/ 3/49 1/ 5/49	....	2/23/49	U	U	Blood: Hb. 12 Gm.; RBC, 4,300,000; WBC, 6,100; N. 40%, L. 52%, M. 2%, E. 3%; serologic reactions negative
26	1/24/40	M	Acute follicular tonsillitis	1 wk.	2	12/29/48 1/ 3/49 1/ 5/49	2/ 9/49 2/16/49 2/23/49	4/ 9/49	U	U	Blood: Hb. 11 Gm.; RBC, 4,300,000; WBC, 8,200; N. 54%, L. 39%, M. 1%, E. 8%; serologic reactions negative
27	2/27/42	M	Chronic hypertrophy of tonsils	1 yr.	1	1/ 5/49 1/ 7/49 1/10/49	....	4/ 9/49	U	U	Blood: Hb. 12 Gm.; RBC, 4,100,000; WBC, 7,500; N. 52%, L. 44%, M. 1%, E. 3%; serologic reactions negative

* G indicates great improvement; M, moderate improvement; S, slight improvement, and U, condition unchanged.
† N. indicates neutrophils; L., lymphocytes; M., monocytes, and E. eosinophils.

REPORT OF ILLUSTRATIVE CASES

CASE 2.—A girl was referred to the nose, ear and throat service of the outpatient department because of large infected tonsils and adenoids associated with a cryptogenic fever. The past history revealed numerous bouts of nasopharyngitis, gastroenteritis, tonsillitis and fever of undetermined origin.

Physical examination showed that the patient was unable to breathe with the mouth closed and nasal obstruction posteriorly from a mass of adenoid tissue. The anterior portion of the nasal chamber was empty. The tonsils were moderately enlarged and there was a slight prominence of the vessels of the postpharyngeal wall. Many anterior and posterior cervical lymph nodes were palpable.

Laboratory Observations: On Nov. 11, 1948, the drop in the sedimentation rate was 30 mm. in forty minutes; on December 16, 20 mm. in forty minutes, and on December 30, 13 mm. in forty minutes. On November 9, the hemoglobin in the blood was 11 Gm. The red cell count was 4,380,000, the white cell count, 4,300, with 42 per cent neutrophils; 49 per cent lymphocytes; 4 per cent monocytes; 2 per cent eosinophils, and 1 per cent basophils. On Feb. 16, 1949, the hemoglobin in the blood was 11 Gm. The red cell count was 3,600,000, the white cell count, 5,800, with 31 per cent neutrophils; 64 per cent lymphocytes; 4 per cent monocytes, and 1 per cent eosinophils. The child was placed under treatment with antireticular cytotoxic serum, and after two series of treatments, no objective or subjective improvement was noted.

CASE 6.—A boy was being treated in the nose, ear and throat service for chronic discharge of the ear of several years' duration, associated with polyp formation in the middle ear. The past history revealed bouts of bronchitis, acute nasopharyngitis, bilateral otitis media and acute tonsillitis. The tonsils and adenoids were removed in 1942. Systemic review during the period of observation indicated nothing abnormal. Physical examination revealed profuse mucopurulent discharge in the external auditory canal on the left, and when wiped away, there was disclosed a large perforation of the drum membrane, through which a polyp protruded. The nose and sinuses appeared relatively normal. There was a moderate amount of lymphoid tissue in the nasopharynx and throat.

Laboratory Observations: A roentgenogram of the chest revealed that the condition of the heart and lungs was within normal limits. On Dec. 3, 1948, the hemoglobin in the blood was 11 Gm. The red cell count was 3,570,000, the white cell count, 5,300, with 57 per cent neutrophils; 39 per cent lymphocytes; 1 per cent monocytes, and 3 per cent eosinophils. On March 27, 1949, the hemoglobin in the blood was 12 Gm. The red cell count was 4,400,000, the white cell count, 4,400, with 42 per cent neutrophils; 49 per cent lymphocytes; 3 per cent monocytes, and 6 per cent eosinophils. Serologic studies of the blood indicated nothing abnormal. In adition to the usual measures applied locally, the child was placed on therapy with antireticular cytotoxic serum, but only slight improvement has been noted to date.

CASE 10.—A girl was first seen in the ear, nose and throat service of the outpatient department on Feb. 26, 1945 because of chronic discharge of the ear of several months' duration and had been attending the clinic intermittently since that time. The past history and systemic review aside from the presenting symptom, indicated no abnormalities. A tonsillectomy and adenoidectomy had been performed in 1943, and in 1947 another adenoidectomy was performed. The patient had been hospitalized several times for acute exacerbations of otitis media with mastoiditis. Physical examination revealed that the nasal septum was

deflected slightly to the left, with some impairment of the airway. The left ear had a profuse mucopurulent discharge filling the external auditory canal and a large central perforation of the drum membrane. The throat was relatively normal in appearance. Lymph nodes were palpably enlarged under the angle of the jaw.

Laboratory Observations: On Dec. 6, 1948, the red cell count was 3,940,000, the white cell count, 5,150, with 63 per cent neutrophils, 28 per cent lymphocytes, 5 per cent monocytes and 3 per cent eosinophils. The hemoglobin in the blood was 12 Gm. On March 2, 1949, the hemoglobin in the blood was 11 Gm. The red cell count was 3,380,000, the white cell count, 6,400, with 53 per cent neutrophils, 42 per cent lymphocytes, 2 per cent monocytes and 3 per cent eosinophils. Serologic studies of the blood indicated nothing abnormal. In addition to the usual measures, the child was placed under treatment with antireticular cytotoxic serum and received one series of injections. She had a local reaction of a large, red, indurated, painful swelling from one injection. For the first time in many years, the ear became dry and has remained so for some time.

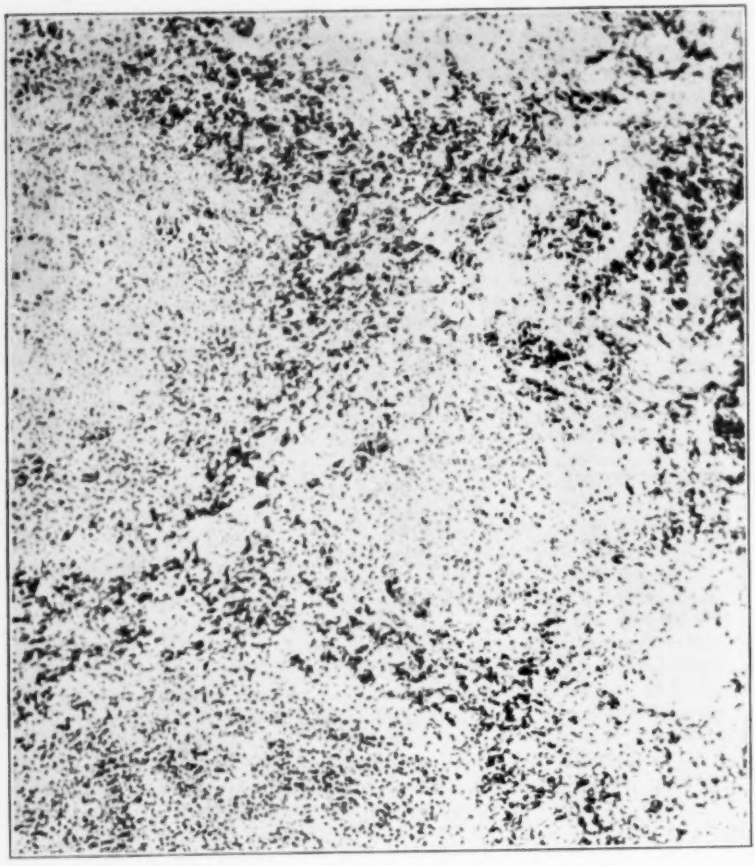
Case 15.—A girl was under the care of the ear, nose and throat service because of chronic discharge of the ear following simple mastoidectomy performed in 1945. The family history and systemic review indicated nothing abnormal. Physical examination revealed diffuse lymphoid hyperplasia of faucial, lingual and nasopharyngeal regions. There was a moderate purulent discharge from the left ear and excessive granulation tissue in the remainder of the middle ear. An audiogram of the right ear at the frequencies of 128, 256, 512, 1024 and 4096 gave a reading of 20 decibels and 10 decibels at the 2048 frequency. The reading of the left ear was 55 decibels at the frequencies of 128, 256, 512 and 4096; 40 decibels for the 1024 frequency, and 50 decibels for the 2048 frequency.

Laboratory Observations: On Dec. 16, 1948, the hemoglobin in the blood was 12 Gm. The red cell count was 3,870,000, the white cell count, 7,700, with 45 per cent neutrophils, 45 per cent lymphocytes, 1 per cent monocytes and 8 per cent eosinophils. On Feb. 25, 1949, the hemoglobin in the blood was 11 Gm. The red cell count was 3,510,000, the white cell count, 5,100, with 52 per cent neutrophils, 40 per cent lymphocytes, 3 per cent monocytes and 5 per cent eosinophils. Serologic studies of the blood indicated nothing significant. A culture yielded Escherichia coli. In addition to local treatment, the patient was placed on a schedule of treatment with antireticular cytotoxic serum and aureomycin, but no amelioration of symptoms or changes were noted on physical examination.

Case 19.—A lad was attending the nose and throat clinic because of recurrent colds and excoriation of both external vestibules of the nose. The past history indicated that he had been seen in other clinics because of numerous episodes of bronchitis, acute follicular tonsillitis and infections of the upper respiratory tract. Physical examination revealed enlarged chronically infected tonsils, adenopathy, bilaterally of the cervical lymph glands, and a small amount of mucopurulent secretion in both sides of the nose. Tonsillectomy and adenoidectomy were performed without any marked changes in the general clinical status. It was decided to give the patient a course of therapy with antireticular cytotoxic serum.

Laboratory Observations: On March 2, 1949, the hemoglobin in the blood was 13 Gm. The red cell count was 4,110,000, the white cell count, 10,500, with 28 per cent neutrophils, 57 per cent lymphocytes, 2 per cent monocytes and 13 per cent eosinophils. A roentgenogram of the sinuses showed thickened mucosa and clouding of the paranasal sinuses, indicating a moderate degree of pansinusitis. Therapy with antireticular cytotoxic serum was given, and during

the first series of injections, an urticarial reaction followed one injection. Examination at completion of the first series revealed the nasal mucosa to be very boggy, with mucopurulent secretion filling both nasal chambers. Nasopharyngeal lymphoid hyperplasia was present. The patient continued to cough at night.



Photomicrograph of section of adenoid tissue in case 20

A second series of injections with this serum was given with no appreciable change noted on physical examination. The patient's mother reported that she believed the child was doing better. Because of pronounced eosinophilia, he was referred to the allergy clinic for further studies.

CASE 20.—A girl was referred by the medical clinic to the nose, ear and throat service of the outpatient department of the Children's Hospital for further observation and treatment of recurrent infections of the upper respiratory tract and related disorders. The past history revealed bouts of colds, acute catarrhal otitis media, nasopharyngitis, bronchitis and tonsillitis since the patient was 5 months old. Physical examination of the nose revealed a moderate amount of mucoid secretion in both sides of the nose and swollen turbinate bodies; the ears were relatively normal; the tonsils were enlarged; the anterior and posterior pillars were congested, and postnasal drip was present.

Laboratory Observations: On Dec. 21, 1948, the hemoglobin in the blood was 10.5 Gm. The red cell count was 3,450,000, the white cell count, 4,800, with 40 per cent neutrophils, 52 per cent lymphocytes and 2 per cent monocytes. On April 8, 1949, the hemoglobin in the blood was 11 Gm. The red cell count was 3,300,000, the white cell count, 7,150, with 46 per cent neutrophils, 50 per cent lymphocytes and 4 per cent monocytes. The patient was given one series of injections of antireticular cytotoxic serum, but no beneficial or untoward reactions resulted, from objective or subjective viewpoints. A tonsillectomy and adenoidectomy were performed, and the pathologic report on the tissue removed follows:

Microscopic Observations: The specimen consisted of sections of adenoid tissue. This tissue was partially covered on one side by mucous membrane. The mucous membrane was desquamated in spots and covered completely by an inflammatory exudate. Deep to the membrane could be seen numerous hyperactive lymph follicles separated by trabeculae of fibrous connective tissue and by invaginations of the mucous membrane. All these structures showed chronic inflammatory changes associated with hemorrhage (incident to operation?) and necrosis.

1930 Chestnut Street.

Dr. Harry Goldblatt, of Cedars of Lebanon Hospital, Los Angeles, supplied the antireticular cytotoxic serum for this study.

SIALOGRAPHY

F. JOHNSON PUTNEY, M.D.
AND
MYRON J. SHAPIRO, M.D.
PHILADELPHIA

DISEASES of the salivary glands have received scant attention in medical literature. Except for tumors and calculi, knowledge of other abnormalities of the salivary glands remains meager. Patients with recurrent pain and swelling of one or both parotid or submaxillary glands require study by other than the usual clinical methods. An additional means of diagnosis, by injecting a radiopaque medium into the duct system and parenchyma of the two chief salivary glands, parotid and submaxillary, is available and should be utilized when the diagnosis is obscure.

Radiopaque materials were injected into Stensen's duct as early as 1904, but the procedure was considered a medical curiosity of no clinical significance until 1926. In that year Carlsten [1] studied normal and pathologic parotid glands roentgenologically, using iodized oil injected into Stensen's duct. Since then many reports have been published, with various attempts to correlate the clinical and roentgen findings.

ANATOMY

Both the parotid and the submaxillary glands are compound racemose glands. The parotid is a purely serous gland; the submaxillary is a mixed gland but is mostly serous.

Parotid Gland.—The parotid gland completely fills and overflows its irregular bed. Several tonguelike parotid lobules extend into the crevices of this mold. One passes forward between the upper part of the ramus of the mandible and the medial pterygoid muscle; another passes upward between the tympanic plate and the capsule of the temporomandibular joint; another passes medially in front of the internal carotid artery to the superior constrictor muscle, where it

From the Department of Bronchoesophagology and Laryngology, Jefferson Medical College, Philadelphia.

Presented before the Section on Otolaryngology of the College of Physicians of Philadelphia, April 27, 1949.

1. Carlsten, D. B.: Lipiodol Injection in Duct of Salivary Glands, Acta radiol. **6**:221-233, 1926.

may be in close proximity to the tonsil. The largest projection is the facial process. It covers the hinder part of the masseter muscle, as well as the temporomandibular ligament and the neck of the mandible, and is prolonged above the parotid duct as the accessory parotid gland. In 50 per cent of cases there are two or more accessory glands.[2]

The parotid duct (Stensen's duct) crosses the masseter muscle a fingerbreadth below the zygomatic arch, turns at right angles around

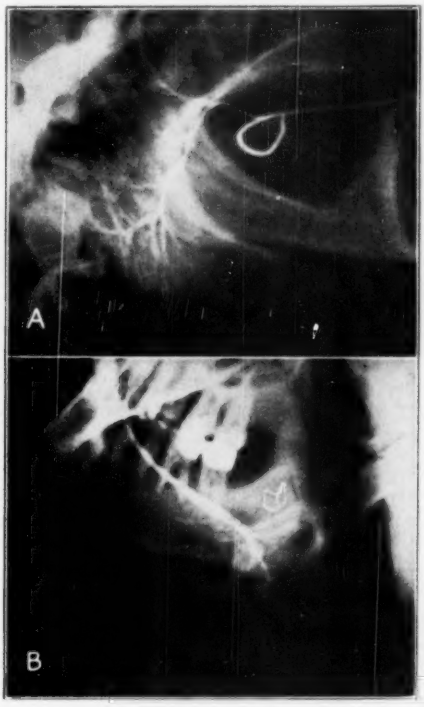


Fig. 1.—Sialogram of (*A*) normal parotid gland and duct and (*B*) normal submaxillary gland and duct.

the anterior border of the masseter and then pierces the buccal fat, buccinator muscle and mucous membrane of the cheek, where its constricted orifice can be seen at the level of the second upper molar tooth.

2. Blady, J. V., and Hocker, A. F.: Sialography: Its Technique and Application in the Roentgen Study of the Parotid Gland, Surg., Gynec. & Obst. **67**:777-787, 1938.

When iodized oil is injected into Stensen's duct, the stream flows along the duct for a distance of ½ to 1 inch (1.3 to 2.5 cm.) and then divides into two branches, one branch going to the accessory lobe and the other continuing to the main gland. As the duct approaches the gland substance, it divides into smaller ducts; these major branches extend into the gland between the lobes and divide still further into minor branches which pass between the lobules and then into the intralobular ducts and finally may reach the acini themselves. Stensen's duct usually varies in direction, and its caliber and direction are influenced by movements of the cheek [3] (fig. 1*A*).

Submaxillary Gland.—The submaxillary (submandibular) gland fills the digastric triangle to overflowing. It extends backward to the parotid gland, from which it is separated by the stylomandibular ligament. It extends upward between the body of the mandible and the mylohyoid muscle and between the insertion of the medial pterygoid and the superior constrictor muscles. It sends a deep process around the posterior border of the mylohyoid muscle along the floor of the mouth as far as the sublingual gland. The duct (Wharton's) leaves this deep portion of the gland and runs across the medial aspect of the sublingual gland, where it lies submucosal in the floor of the mouth. It opens beside its fellow at the apex of the caruncle, which appears at the root of the frenulum of the tongue.

When fluid is injected into Wharton's duct, the stream divides, as does that going to the parotid gland, into major and minor branches and finally enters the acini (fig. 1*B*).

TECHNIC OF INJECTION

Preliminary roentgen studies of the soft tissues of the affected gland should be made. The sialogram is made in the x-ray room so that roentgenograms can be taken immediately after injection. Preferably one gland is studied at a time. The injection is relatively painless, but topical anesthesia may be used in the region of the orifice of the duct.

The duct is usually easily located. If there is difficulty, massaging of the gland may cause saliva to issue from the duct. If this is not successful, having the patient suck a piece of lemon or drink some other sour liquid will usually demonstrate the orifice. Painting the area with a colored solution or blowing air on the duct may disclose the opening. Once located, the orifice is dilated with a lacrimal dilator,

3. (*a*) Hobbs, W. H., and Sneierson, H.: Infections of the Parotid Gland, Am. J. Surg. **32**:258-271, 1936. (*b*) Kimm, H. T.; Spies, J. W., and Wolfe, J. J.: Sialography, with Particular Reference to Neoplastic Diseases, Am. J. Roentgenol. **34**:289-296, 1935. (*c*) Pancoast, H. K.; Pendergrass, E. P., and Schaeffer, J. P.: The Head and Neck in Roentgen Diagnosis, Springfield, Ill., Charles C Thomas, Publisher, 1940.

graded whalebone filiform bougies or similar dilators. Kimm and associates [3b] stated that they slit the caruncle, if necessary, to cannulate Wharton's duct. Various types of cannulas are used, but any small blunt cannula will suffice. Ordinary hypodermic needles, 20 to 26 gage, with a blunt tip, or lacrimal needles are satisfactory. Payne [4] recommended using a glass medicine dropper with a thin drawn-out tip. We have been using a short piece of no. 4 ureteral catheter, which can be introduced into the duct without difficulty and may be left in position for a considerable time without discomfort to the patient.

Iodized oil is the most satisfactory radiopaque agent. Sodium iodide has been used, but it is irritating. Other water-soluble opaque agents pass too rapidly into the parenchyma. To reduce its viscosity, the iodized oil is warmed or diluted with liquid petrolatum.

The cannula is introduced 1 to 1.5 cm. into the duct. When the contrast medium is injected into the parotid duct, pulling the cheek outward and upward will tend to straighten the initial bend of the duct and allow the cannula to be passed more readily. Slow injection under little pressure is essential, as distention of the ducts causes discomfort. One to 1.75 cm. can be injected into a normal parotid duct system. When there is dilatation of the ducts, more can be injected. The submaxillary duct system will hold almost the same amount. After the injection, the opening of the duct is compressed with a piece of gauze to prevent backflow, and roentgen exposure is made immediately. Pancoast, Pendergrass and Schaeffer,[3c] Payne [4] and others have recommended the taking of two sets of roentgenograms: one with a small amount of iodized oil injected into the ducts and the second with a larger amount of iodized oil. The first set is to demonstrate the duct system, and the second, the parenchyma. With use of a ureteral catheter which is left in the duct, 0.5 cc. of iodized oil is injected and exposures are made. Immediately, an additional 1.00 cc. of iodized oil is injected, and a second set of roentgenograms is made. This technic has an additional advantage in that the catheter, left in place, prevents backflow.

Generally there is little reaction. Some patients complain of a feeling of distortion or discomfort for a few hours. There may be a slight, short-lived swelling of the gland, particularly in cases of recurrent parotitis.

ROENTGENOGRAPHY

A. Parotid Gland.—At least two roentgenograms are necessary: (1) a lateral projection with the head extended, to avoid superimposition of the parotid shadows on the cervical portion of the spine, and (2) an anteroposterior view, which is especially useful in case of tumors.

4. Payne, R. T.: Sialography: Its Technique and Application, Brit. J. Surg. **19**:142-148, 1931.

Some authors[5] recommend (3) an oblique (mandibular) and fronto-submental view.

B. Submaxillary Gland.—The affected side of the face is placed against the cassette, and the opposite side of the mandible is thrown slightly upward. The shadow of the injected duct and the submaxillary gland is then seen superimposed on or just below the mandible on the side being studied. Two exposures are made: (1) an anteroposterior view and (2) an occlusal view to show the anterior part of the duct.

The injection may be done under the fluoroscope, but the shadows are difficult to see in the small area being examined. Normally the injected gland will eliminate the opaque material in two to forty-eight hours. It is eliminated more slowly and may take two or three weeks if the gland is diseased, if the injection is repeated or if the parenchyma was filled.

INDICATIONS

Sialography is indicated (1) to demonstrate the relationship of the normal duct and gland to adjacent structures; (2) to demonstrate salivary fistulas; (3) to demonstrate calculi, foreign bodies, strictures and diverticula of the ducts; (4) to demonstrate and confirm a clinical diagnosis of chronic recurrent parotitis; (5) to investigate possible involvement of the gland and the ducts in a variety of pathologic conditions affecting the cheek, parotid region or upper part of the neck; (6) to determine whether a tumor is an encapsulated growth in the gland substance, a tumor infiltrating the duct system and gland or a mass extrinsic to the glands and ducts proper; (7) to determine the proximity of the pharyngeal process of the parotid gland to a salivary gland tumor in the tonsillar region; (8) to aid in the planning of an operative procedure in the parotid area by outlining the depth of a glandular tumor, and (9) to investigate the structure and function of the salivary glands in cases of dry mouth (the emptying time of the gland may be of significance).

In cases of chronic stone formation, after the stone has been removed, sialectasis due to continued chronic inflammation of the duct may be present; sialography is indicated in such cases. It is also indicated in the case of patients complaining of a foul taste in the mouth when other causes have been eliminated.

CONTRAINDICATIONS TO SIALOGRAPHY

Sialography is contraindicated in the presence of acute infection in the gland.

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5. (*a*) Shuls, M. D., and Weisberger, D.: The Sialogram in the Diagnosis of Swelling About the Salivary Glands, S. Clin. North America **27**:1156-1161, October 1947. (*b*) Blady and Hocker.[2] (*c*) Kimm, Spies and Wolfe.[3b]

In some cases of chronic parotitis injection of the duct may cause a temporary slight exacerbation. When a biopsy is anticipated, sialography should be done first because the biopsy site may simulate a fistula causing puddling of the iodized oil and obscuring details of the ducts.

SIALOGRAMS IN DISEASES OF THE SALIVARY GLANDS

Most of the studies that have been reported[6] have been concerned mainly with the changes which occur in sialograms of the parotid gland. Perhaps this is partly due to the greater difficulty of injecting contrast medium into the submaxillary gland and partly because the parotid gland, in comparison with the submaxillary, is affected by more diverse and obscure pathologic conditions.

Obstructed Duct.—Obstruction of the duct may have the following causes: 1. Recurrent parotitis due to mild persistent infection of the duct. Similar infections occasionally affect the submaxillary gland. The

Fig. 2.—Sialogram of obstructed submaxillary duct, revealing marked dilatation of the main duct due to an extrinsic tumor.

obstruction is caused mainly by swelling of the duct and inspissated secretion. 2. Infection of the gland with destruction and/or obliteration of the ducts. 3. Calculi. 4. Trauma to the duct or gland. 5. Strictures —congenital or acquired.

The sialographic changes are the following: 1. Dilatation of the main duct and the terminal portion of its chief contributory ducts. This is due to narrowing of the duct near the orifice (fig. 2). 2. Slight to marked dilatation of the peripheral ducts (sialectasis or sialangectasis).

6. Anthony, D. H., and Fisher, D. F.: Diseases of the Salivary Glands and Their Ducts, J. Tennessee M. A. **41**:362-377, 1948. Barsky, A. J., and Silberman, H.: Roentgen Visualization of the Parotid Gland by Means of Lipiodol Injection, Ann. Surg. **95**:46-51, 1932. Hobbs, W. H.; Sneierson, H., and Faust, C. L.: Acute and Chronic Infections of the Parotid Gland, Surg., Gynec. & Obst. **54**:555-563, 1932. Pyrah, L. N.: Chronic Parotitis: A Report of Four Cases with Sialograms, Brit. J. Surg. 508-515, 1933. Blady and Hocker.[7] Kimm, Spies and Wolfe.[8b] Shuls and Weisberger.[8a]

This picture may be simulated by a normal gland in which some iodized oil has passed into the parenchyma, appearing as globules (fig. 3). 3. Incomplete filling of the ducts. This may be caused by one or more calculi causing obstruction to the main duct or smaller ductules. Incomplete filling is also caused by fibrosis atrophy of the gland from long-standing infection.

Tumors.—The changes that occur depend to a certain degree on the type of tumor involvement. All tumors—infiltrating, encapsulated, intrinsic or extrinsic—cause pressure changes in the duct system and obliteration of the fine ductules. It is the distribution of these changes, that are helpful in diagnosis of tumor.

Fig. 3.—Sialectasis of parotid gland; *A*, anteroposterior view; *B*, lateral view.

Fig. 4.—Submaxillary defect from intrinsic tumor. On the sialogram a shadow defect is evident in the anterior portion of the submaxillary gland. The patient had metastatic tumor of the cervical lymph nodes which compressed the submaxillary gland.

The roentgenographic shadows of the parenchyma should be studied as carefully as those of the ducts.

Extrinsic tumors in the vicinity of the gland cause a defect in the glandular shadow, frequently associated with inability to visualize, or partial visualization of, the smaller ducts in the affected area. The margin of the gland is usually well demarcated, and this, together with a uniform radiodensity of the parenchyma, signifies a secondary effect due to pressure (fig. 4).

Usually in the presence of a benign tumor of a salivary gland a filling defect is outlined by displaced ducts and changes in the parenchy-

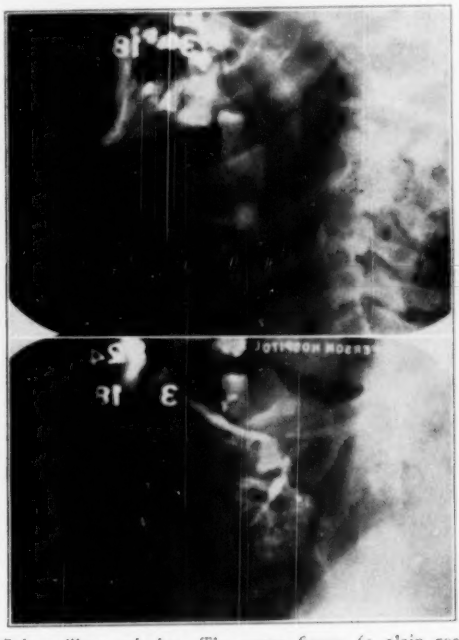

Fig. 5.—Submaxillary calculus. The upper figure (a plain roentgenogram) reveals a calculus in the region of the submaxillary gland. By sialographic study the calculus was localized in the posterior portion of the duct before its entrance into the gland.

mal shadow. There is no sharp line of demarcation between the tumor and the normal gland. Sialectasis, generalized or localized, may occur beyond the tumor. If iodized oil enters the tumor area, abnormal shadows are produced. The tumor may press on the duct or displace the gland forward, causing buckling of the duct. The location of the tumor in relation to the rest of the gland and the main duct helps in the planning of a suitable surgical approach.

Malignant tumors of the salivary glands are difficult to differentiate from benign tumors. Kimm and associates [3b] stated that there are no real criteria for malignancy. Blady and Hocker [2] found changes which were considered infiltrative in 62 per cent of cases of proved carcinoma. These changes include (1) irregular defects in the gland parenchyma; (2) distortion of the normal duct alinement; (3) incomplete filling of, irregularity of, and failure to visualize, the duct system; (4) irregular puddling in the tumor area due to destruction of ducts with escape of iodized oil into the tissues; (5) complete filling distal to the tumor unless the main duct was interfered with by the tumor.

Salivary Calculi.—The diagnosis of salivary calculi is usually simple, but small calculi, especially in the parotid gland, are sometimes missed by plain roentgen examination. Sialograms demonstrate the site of the obstruction and the secondary changes in the duct. By this method intraglandular calculi can be distinguished roentgenographically from

Fig. 6.—Accessory parotid gland. The patient complained of intermittent swelling in the parotid region. An accessory parotid gland was revealed by the sialogram.

those lying within the ducts (fig. 5). In salivary fistulas this procedure helps in determining whether the fistula connects with a large duct or an outlying lobule.

Multiple abscesses produce large irregular cavities which are filled through the ducts. In Mikulicz [1] disease and sarcoid, no changes in the duct patterns have been described. Accessory salivary glands sometimes are symptomatic, producing pain from excessive secretion not adequately removed by the duct (fig. 6).

CONCLUSIONS

Sialography is an aid in the diagnosis of diseases of the salivary glands. Alone, the procedure may be of little significance, but as an adjunct to clinical observations it can be of great help in solving many diagnostic problems.

ACUTE SUBDURAL EMPYEMA SECONDARY TO FRONTAL SINUSITIS

Report of a Case With Recovery

JOSEPH A. MUFSON, M.D.
AND
MARVIN WAGNER, M.D.
MILWAUKEE

AMONG the intracranial complications of frontal sinusitis, subdural empyema is perhaps the least common. Although the lesion has been recognized post mortem for more than fifty years, it has been only within the past few decades that clinical reports with suggested methods of treatment have appeared in the literature. Prior to the advent of the sulfonamide drugs and penicillin, cases of subdural empyema almost uniformly ended with death. With the aid of these new antibacterial agents, neurologic surgeons are now obtaining increasingly better results with all intracranial infections, and reports of cases of subdural empyema with successful treatment are beginning to appear in the literature.

In the past, subdural empyema had been identified by a variety of names, such as cortical abscess, circumscript meningitis, internal purulent pachymeningitis and phlegmonous meningitis. Kubic and Adams,[1] in their excellent review of the subject, have adopted the term "subdural empyema" as more appropriate, since the collection of pus is not circumscribed, as in a true abscess, but lies in a preformed space comparable to the pleural cavity.

Courville and Nielsen [2] have adequately discussed the spread of the infection from the frontal sinus to the subdural space. They stated that the infection travels from the mucosa of the sinus through perforating veins in the posterior wall of the sinus to gain access to the dura. The process may then proceed with thrombophlebitis of the dural veins, with or without thrombosis of the superior longitudinal sinus, and retrograde thrombosis of the afferent superior cerebral veins. Such involvement would explain development of subdural infections without destruction of the sinus wall or dura.

From the Department of Surgery, Marquette University School of Medicine and Department of Neurosurgery, Mount Sinai Hospital.

1. Kubic, C. S., and Adams, P. D.: Subdural Empyema, Brain **66**:18-42, 1943.

2. Cited by Kubic and Adams.[1]

In the past, treatment of subdural empyema has been attended by a high mortality rate (Courville and Nielsen,[2] 100 per cent; Kubic and Adams,[3] 85 per cent; Kahn,[3] 83 per cent, and Ray and Parsons,[4] 100 per cent). For this reason it was thought worth while to report a case with successful treatment in order to emphasize the fairly constant clinical features which subdural empyema presents.

REPORT OF CASE

M. K., a female clerk aged 29, was admitted to the hospital on May 6, 1948. Her illness began with an infection of the upper respiratory tract three weeks prior to her admission to the hospital. Because of pronounced nasal symptoms, an otolaryngologist removed a polyp and a portion of the middle turbinate body from the right nostril. Following this procedure, severe headaches developed in the right frontal region. Her family noted that she began to have lapses of memory and appeared incoherent at times. One week prior to her entry to the hospital, the patient experienced difficulty in using her left hand. Her temperature became elevated, reaching 104 F. on May 3. She began to vomit repeatedly and continued to complain of severe headache.

Examination revealed a very lethargic patient, who was able to obey commands slowly. There was tenderness over the region of the right frontal sinus but no swelling of the scalp or eyelids. There was a supranuclear weakness on the left side of the face and mild hemiparesis on the left. The tendon reflexes were slightly exaggerated on the left side, but Babinski's sign was not elicited. The abdominal reflexes on the left were diminished as compared to those of the right. There was mild nuchal rigidity. The optic fundi were normal. The temperature was 99.6 F. orally, and the pulse rate was seventy per minute.

The red cell count was 4,130,000 and the hemoglobin 76 per cent. The white cell count was 40,130, with 80 per cent polymorphonuclear leukocytes, 14 per cent stab cells and 6 per cent lymphocytes. The urine was normal except for a trace of albumin. The spinal fluid was clear and colorless and contained 550 cells, which were predominantly polymorphonuclear leukocytes. Roentgenograms of the skull revealed clouding of the right frontal sinus.

Within twelve hours after admission to the hospital, the patient became increasingly comatose, and there was a definite increase in the hemiparesis on the left. The right pupil became dilated and fixed to light. The optic disks were flat. The patient began to exhibit jacksonian convulsions, involving the left arm and the left side of the face. On May 7, fourteen hours after her admission to the hospital, the patient was taken to the operating room, where a burr hole was made in the right frontal region 4 cm. above the eyebrow. The dura was opened, and, immediately, thin, brownish, turbid fluid issued from the subdural space and was followed by green, creamy pus. The opening in the skull was enlarged with rongeurs, and when the brain was carefully depressed, an additional amount of pus was evacuated

3. Kahn, E. A.: Brain Abscess, in Bancroft, F. W., and Pilcher, C.: Surgical Treatment of the Nervous System: Cranial and Intracranial Surgery, Tumors, Epilepsy and Cranial Nerve Disorders, Spinal Cord, Autonomic Nervous System, Peripheral Nerves, Philadelphia, J. B. Lippincott Company, 1946, pp. 88-90.

4. Ray, B. S., and Parsons, H.: Subdural Abscess Complicating Frontal Sinusitis, Arch. Otolaryng. **37**:536-551 (April) 1943.

with suction. A rubber catheter was then placed into the subdural space down to the region of the frontal sinus and was brought out through the wound to act as a drain.

Postoperatively, the patient continued to exhibit convulsions on the left for seventy-two hours. These were controlled with anticonvulsants. She was given 300,000 units of duracillin® daily, and 50,000 units of penicillin was administered intramuscularly every three hours. Sodium sulfadiazine (5 Gm.) was given intravenously on May 9 and May 10, and then 1 Gm. of sulfadiazine was given orally every four hours. Immediately following operation, the patient began to improve, but her state of consciousness was impaired by the repeated convulsive

Fig. 1.—Posteroanterior view of skull. *A* indicates the location of the trephine opening used to drain the subdural empyema; *B*, the mottling of the frontal bone indicative of an osteomyelitic process.

seizures. There was a moderate amount of drainage from the wound for a few days, and during this time the catheter was irrigated with a solution of penicillin containing 10,000 units per cubic centimeter. By May 12 the patient was conscious and quite alert. Slight weakness of the left arm was still demonstrable. The temperature, which had risen to 102 F. after operation, returned to normal by May 10 and remained normal thereafter. The drain was removed on the seventh postoperative day, and it was noted at that time that the patient was becoming increasingly drowsy. On May 15 she became deeply stuporous and could barely be aroused. Despite the absence of papilledema or an increase in the hemiplegia, it was believed that the patient might be harboring an abscess in a lobe in the right frontal region, in addition to the subdural infection.

She was, therefore, taken to the operating room again, and the original wound was reexplored. There was no reaccumulation of pus in the subdural space, but the brain appeared to pout slightly through the bony defect. After thorough coagulation of one point on the surface of the brain, a cannula was inserted into the right frontal lobe, but nothing unusual was encountered. The wound was closed tightly, and penicillin therapy was continued. The patient remained stuporous for an additional period of forty-eight hours, but during this interval the temperature and blood pressure remained normal. By May 17 she began again to respond, and from that point on the course was definitely one of improvement. The penicillin was stopped on June 2. The patient was allowed to get out of bed on that day and was discharged from the hospital on June 6.

Two weeks after the patient's discharge from the hospital, a swelling developed over the midfrontal region and gradually increased in size. Roentgenograms of

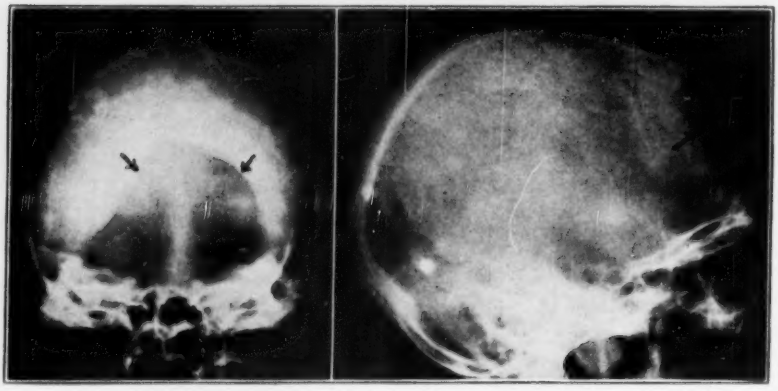

Fig. 2.—Front and lateral views, demonstrating the extent of removal of bone necessary to eradicate the osteomyelitis.

the skull (fig. 1) disclosed a focus of osteomyelitis of the frontal bone. Accordingly, the patient was readmitted to the hospital, and on July 3 the frontal bone was exposed through a coronal incision. A large subgaleal abscess was encountered and evacuated. The underlying bone was soft and necrotic. It was removed piecemeal with rongeurs until healthy-appearing bone was visible on all sides. The frontal sinus was entered, and both the anterior and the posterior walls were removed, together with chronically infected mucous membrane. With all osteomyelitic bone removed (fig. 2), the extradural region was drained, through both the nasofrontal duct and the lateral angles of the coronal incision. Penicillin and sulfadiazine, which had been given preoperatively, were continued during the postoperative period. The convalescence was smooth. The patient was ambulatory on the fourth postoperative day and was discharged from the hospital on July 16. The operative wound was completely healed. Her convalescence at home has been uneventful. She was to return after an interval of six months for cranioplasty.

COMMENT

Acute subdural empyema is unquestionably a disastrous complication of paranasal sinus infection. Most of the cases reported in the literature prior to 1948 are studies made on autopsy. In many cases death occurred because intracranial exploration was delayed while the focus of infection in the sinus or mastoid was attacked. A review of the salient clinical features of the cases reported in the literature reveals that acute subdural empyema presents a fairly constant course. The illness begins with an acute frontal sinusitis or an acute exacerbation of a chronic sinusitis. The first indication of involvement of the subdural space may be a rapidly developing hemiparesis or a jacksonian convulsion on the contralateral side. This is almost constantly associated with a decided rise in temperature, and the patient appears critically ill. With the appearance of focal neurologic signs, the course is rapidly downhill. Evidence of meningeal irritation may or may not be pronounced, and the spinal fluid merely shows signs of mild irritation.

The differentiation of subdural empyema from intracerebral abscess is all-important, once focal neurologic signs make their appearance. With abscess, the tendency is to delay surgical treatment as long as possible to allow for encapsulation. Such delay would be disastrous in the case of subdural empyema, which requires early and adequate drainage of the subdural space. As a rule, the initial stages of cerebral abscess are milder and insidious, and the course of the illness is more drawn out. The temperature is seldom high and may even be normal. Acute subdural empyema, on the other hand, presents all the features of an acute fulminating infection with high fever, prostration, leukocytosis and rapidly developing coma.

The case presented in this report clearly emphasizes this fairly constant sequence of events. It illustrates further the importance of immediate operation once the diagnosis is suspected. In our case, there was rapid progression from light stupor to deep coma within a period of twelve hours; in fact, at the time of operation, the patient looked moribund. Surgical drainage of the subdural space may be carried out through one enlarged burr hole or through multiple burr holes. The important thing is to provide adequate drainage. As in any intracranial bacterial infection, operation should be combined with intensive antibiotic and sulfonamide therapy. Surgical procedures to eradicate primary foci of infection in the paranasal sinuses and to remove osteomyelitic bone, as in the case presented here, should be done as soon as feasible, but exploration of the subdural space should never be delayed. Once the infection has entered the subdural space, these foci are of secondary importance and can wait until the subdural collection has been evacuated.

There can be no doubt that penicillin and the sulfonamide drugs have greatly altered the course of all intracranial infections, and the

increasing reports of cases of subdural empyema with successful treatment make it clear that even this treacherous complication of acute sinusitis can be attacked with remarkable success.

One must remember that intracerebral abscess may accompany or even follow acute subdural empyema. This complication is to be suspected if a relapse develops following a period of improvement after drainage of the empyema. Webster and Gurdjian [5] have recently presented 2 cases of intracerebral abscess associated with subdural empyema. The abscesses were suspected because of relapse, and this led to diagnostic pneumoencephalographic studies. Because of the successful outcome of their 2 cases, these authors advocated routine pneumoencephalography as a final step in the program of treatment of acute subdural empyema. They state that "when this study is normal, the patient may be discharged with assurance that he is in all probability cured." While we believe that routine air studies may not be necessary in all cases, one should not hesitate to employ pneumoencephalography if there is any doubt of complete irradication of the suppurative lesion.

5. Webster, J. E., and Gurdjian, E. S.: Acute Subdural Abscess, S. Clin. North America **28**:1289-1297, 1948.

BASE OF THE NOSE

Anatomy and Plastic Repair

BRUNO L. GRIESMAN, M.D.
NEW YORK

DEFORMITIES of the base of the nose are frequently combined with other nasal malformations. The size and direction of the nostrils, the thickness and contour of the alae, the configuration of the columella, the breadth and height of the lobule and the form of the alar-facial junction are problems of plastic repair for the surgeon. Occasionally it is their correction which decides the final result. The nares are not mere openings for the entrance or escape of air but are functioning organs from a physiologic point of view. They are essential elements in the production of facial expression.[1]

By looking at a collection of photographs of nasal bases it seems rather difficult to find two persons with similar nasal bases, just as it it unusual to discover two persons with matching external noses. Only identical twins may make an exception to this rule.

I shall enumerate briefly a few anatomic facts regarding the base of the nose for the better understanding of the operative procedure.

The nasal base shows a rather complex construction. It is made up of several structural members: (1) the tip, (2) the lobule, (3) the columella, (4) the alae, (5) the alar-facial junction, (6) the nasolabial ridge, (7) the nostrils and (8) the subnasal point (fig. 1). The columella should always project downward from the plane of the two wings of the alae. If this is not the case, the base presents the undesirable sight of a retracted columella. In the schematic diagram (fig. 2) three horizontal lines are drawn: (1) *ab*, through the most prominent point of the tip; (2) *cd*, through the upper (ventral) rim of the apexes of the nostrils, and (3) *ef*, through the posterior lower (dorsal) rim of the nostrils. One will notice from these lines that the height of the base is made up by two formations: (1) the height of the lobule *XY* plus (2) the height of the columella *YZ*. The line *XY* is bisected at *V*, and perpendicular to it a horizontal dotted line is drawn through the point of bisection. The extension of the breadth

From the Department of Otolaryngology, New York Polyclinic Medical School and Hospital.

1. Webster, G. V.: Plastic Surgery of the Nares, California Med. **67**:302-308 (Nov.) 1947.

of the lobule is clearly defined by the intersection of the dotted line with the outline of the nose. Abel,[2] who tried to measure the different parts of the base, concluded that in women growth of the lobule stops at the age of 16 years, in contrast to men, in whom the lobule continues to enlarge as age progresses.

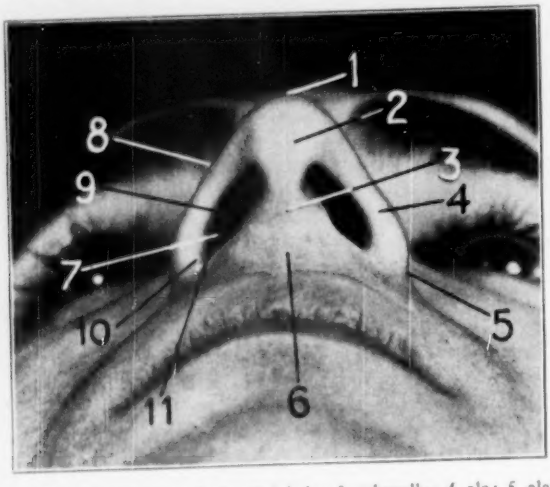

Fig. 1.—Base of the nose; *1*, tip; *2*, lobule; *3*, columella; *4*, ala; *5*, alar-facial junction; *6*, nasolabial ridge; *7*, nostril; *8*, outer border of the ala; *9*, inner border of the ala; *10*, foot of the ala; *11*, alar-facial groove.

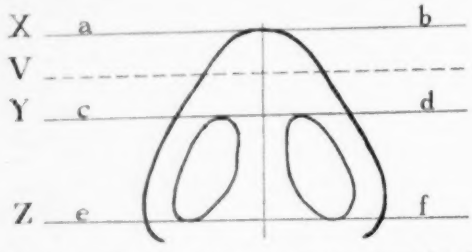

Fig. 2.—Basal view. The height of the base (*xz*) is the sum of the height of the lobule (*xy*) and the height of the columella (*yz*). Further details are given in the text.

The width across the apexes of the nostrils is approximately three quarters of the width of the base of the nose at the level of the

2. Abel, W.: Die Vererbung von Antlitz und Kopfform des Menschen, Ztschr. f. Morphol. u. Anthropol. **33**:261-345, 1934.

alar-facial junction.[3] The ventrodorsal length of the lobule is one third that of the columella. The nostrils should converge toward the tip and form an angle, which may differ individually from 30 to 115 degrees or more. The shape, direction and size of the nares are simply functions of the surrounding structures, i. e., a straight or wedge-shaped columella, the development of the lobule, the thickness (fleshiness) or curvature of the alar wings and, last but not least, the alar-facial junction. Long nostrils may be present because of a long and wide columella and an atrophic lobule. Large nostrils originate from a thin columella and flaring alar wings. Small nostrils are present because of atrophic lower lateral cartilages, thickness of the alar wings, short columella and hypertrophic lobule. Slitlike nares are formed by hypertrophic mesial crura of the lower lateral cartilages and a wide collumelar base. Round nostrils are caused by a hypertrophic wide lobule, convex alae and a wide and short columella.

Asymmetric nares are frequently combined with a deflection of the nasal septum.

Breadth and length of the nostrils depend on many factors. The length goes hand in hand with the enlargement of the entire nose. Abel [3] stated that boys before their thirteenth year of life have nostrils of smaller width than girls of the same age. During the later years of life this condition is reversed. The point of interest is that the direction of the nostrils is probably significant for purposes of testing food by smell.[4] Position, size, dilation and choking of the nostrils are important physiologic factors for the quantity and quality of nasal breathing.

If one looks at the base of a nose during forced respiration, one will notice that the maximal dilation takes place near the alar-facial junction. The cartilaginous part of each naris is not noticeably widened. The lobule and columella seem to brace themselves against the stress of the dilating muscles.[5]

Once the physiologic requirements of the alae are understood, the necessary plastic surgery can be done without interference with them.

THE ALAR-FACIAL JUNCTION

The junction of the alar wing with the upper lip varies individually. By studying a photograph (fig. 1) of the base of the nose one will observe that the alar wing is represented by an outer margin (*8*) and an inner rim (*9*). The inner line (*9*) is clearly distinguished as the lateral border of the nostril. The particular part of the alar column which

3. Fomon, S.; Silver, A. G.; Gilbert, J. G., and Syracuse, V. R.: Physiologic Surgery of the Nares, Arch. Otolaryng. **39**:608-629 (May) 1948.

4. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929, p. 62.

5. Griesman, B. L.: Effect of a Typical Nasal Plastic Correction on Facial Expression, Arch. Otolaryng. **46**:624-643 (Nov.) 1947.

joins the upper lip is called the foot (*10*). In most cases a kind of a groove (*11*) can be seen which marks the line of connection between the sole of the foot (*10*) of the wing and the region of the upper lip (*6*).

Distinct anatomic peculiarities can be observed by comparing the different forms of alar-facial junction. It seems practicable to bring the great variety of individual types of alar-facial junctions under three

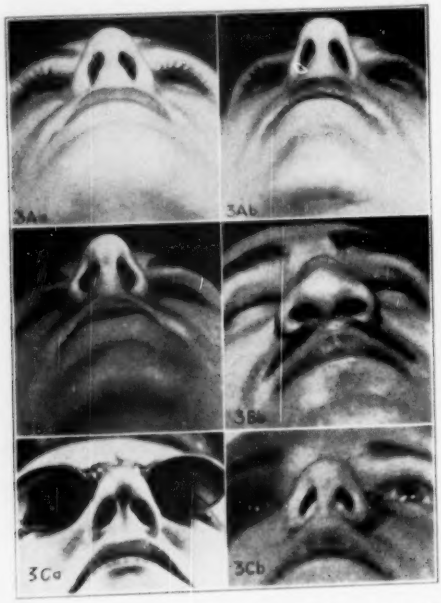

Fig. 3.—The three types of alar-facial junction. *Aa* and *Ab*, cheeked junction; *Ba* and *Bb*, labial junction; *Ca* and *Cb*,[2] columellar junction. *Ab*, *Ba* and *Bb* are from photographs belonging to S. Fomon; *Cb* from a photograph belonging to W. Abel.

classifications according to the specific part of the base on which the foot rests.

The first class presents the "cheeked junction." Here the foot of the alar shaft appears absorbed laterally into the cheek in an almost straight line. It seems as if the line of the outer border of the alar column runs directly into the cheek, independent of the thickness of the ala. The foot may stand nearly perpendicular or slightly bent. A line of demarcation designating the junction is frequently present.

Figure 3 *A* demonstrates this type: *Aa* shows a wide base of the columella and a thickened foot of the alar wings; *Ab* shows a flat nasal tip, a hypertrophic lobule and a wide base of the columella.

The second class is described as the "labial junction." Here the foot of the wings has a tendency to turn medially away from the cheek to find its place of junction at the medial part of the upper lip. Figure 3 *Ba* shows the foot of the wings with a clearly visible curvature; figure 3 *Bb* shows flaring nostrils presenting a definite turning toward the upper lip.

The third class is characterized by the "columellar junction." In this junction the lines of the outer and inner margin of the alar wings (*8* and *9*) do not end in a footlike manner, as shown in the former photographs. Instead, the foot stretches and extends into a tubule several millimeters in length which runs along the inferior and median border of the nostril in an unbroken sweep. It appears attached to the side of the columella by taking an upward course. The tubule varies greatly in length, diameter and shape. This excess of the columella should not be mistaken for the flare of the mesial crura; their end pieces sometimes project into the nares and gives the columella a similar appearance.

Figure 3 *C* demonstrates two types of columellar junction: In *Ca* the alae seem to meet the base of the columella and are drawn across the lower circumference of the nostril; in *Cb* the foot extends in a loop-shaped manner semicircularly along half of the length of the columella.

Difference in age does not play any role in the design of the alar-facial junction. Children as young as 2 or 3 years old show a distinctly developed individual variation of one of the three principal groups of alar-facial junction. It is not surprising that identical twins demonstrate an amazing similarity in their alar-facial junction. Injuries of the nose during ontogenetic development do not exercise any influence on the definite form of the junction (Abel [2]).

THE LOWER PROFILE WIDTH

When a study of the nasal base is made, one should always consider a basal view photograph in relation to a profile photograph. By this method anatomic variations can easily be detected. Figure 4 presents the linear dimensions of a profile photograph of the nose. A line (*xy*) is drawn from the tip (*T*) to the alar-facial junction (*D*). This line is called the lower profile line. The nasal tip, lobule and columella project forward from the subnasal point (*S*). The posterior part of the nostril and of the wing of the ala and the alar-facial junction extend dorsally (posteriorly) from point *S*. The alar-facial groove (*G*) is a genuine fold in the skin designating the posterior margin of the ala against the cheek.

By drawing a horizontal line (*AB*) through the subnasal point (*S*) and two perpendicular lines from the nasal tip (*EF*) and the alar-facial groove (*CD*) to the line *AB*, one can measure the distance *DF*, i. e., the lower profile width. As can be seen in figure 4, it consists of several components: (1) a forward projecting part of the external nose (*SF*) formed by (*a*) the columella, plus (*b*) the lobule, and (2) the backward projecting part of the alar-facial junction (*SD*), i. e., the distance of the alar-facial groove from the subnasal point (*S*). The profile photograph shows the height of the ala, its position with reference to a horizontal line and the direction, depth and curvature of the alar-facial groove. It is worth while to bear in mind that the length of the nostril shows a remarkable tendency to increase during the years of growth.

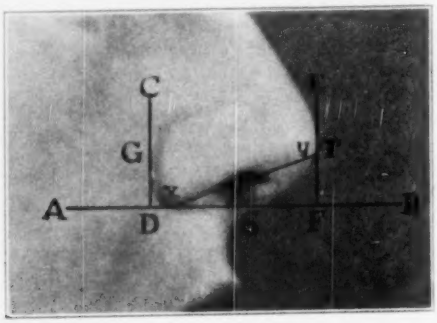

Fig. 4.—Photographic analysis of the nasal base in profile view. Leonardo da Vinci pointed out that the length of the profile line *xy* is equivalent to the width of the nasal base from the attachment of the ala of one side to that of the other side. Further details are given in the text.

STANDARD SURGICAL PROCEDURES

The technic of incisions around the alar-facial junction consists in a variety of procedures to accomplish widely varying aims. They are equally applicable for the improvement and the transformation of unsatisfactory shapes and sizes of the alar wings, the alar-facial junction and the nostrils.

In order to make the principles of surgical procedures more easily understandable, I have classified the incisions into two main groups: (1) the marginal incisions and (2) the encircling incisions. The term "marginal" is used to indicate that the incision extends actually into the margin of the nostril.

A marginal incision can be performed (*a*) with resection of tissue from the alar wing or alar-facial junction or rim of the nostril or (*b*) without resection or sacrifice of any tissue from these regions. The latter type of incision is preferred on patients with small nostrils with

an abnormally wide or short columella. The reason for this is that if the alar wing is not severed along its junction any excision of subcutaneous tissue from the wide columellar base or a submerged suture to correct the shortness of the columella would exert a disfiguring pull on the alae. In such cases the alae are simply separated along their junction fold and reunited to a narrowed or lengthened columella by atraumatic sutures without any loss of alar tissue.

In contrast to the "marginal" incisions are the "encircling" small excisions of skin and subcutaneous tissue for the approximation and taking in of the alae. These incisions do not extend into the margin of the nostrils but they help to narrow the wide distance between the two alar junctions. Atraumatic sutures are used to close the small skin defects.

The outer periphery of the nasal base is shaped like a parabola. The wings of the alae present an outer and inner rimmed line (figs. 1, 8 and 9). Occasionally it may be sufficient for plastic repair to rectify only one of these two lines. In other cases both rims require correction.

Most authors [6] agree on the feasibility of changing the shape, the direction, the position, the junction and the size of the alae by excision of a great variety of geometric forms from the base of the nostrils, from the alar-facial junction or from the alar wings itself. This excision can be performed either intranasally or through the full thickness of the wings.

Here the question arises which type of operation should be applied in individual cases—which special geometric form best restores a graceful contour of the base without impeding the breathing space. The whole ala or a part of it can be shifted around, the nostrils narrowed, widened or changed in direction and the base of the columella and the foot of the alar-facial junction thinned. The type of excision has to be chosen according to the expected effect on the junction and on the nostrils.

There are five principal geometric forms to be considered (fig. 5):

1. A triangular prism or wedge-shaped section with the apex inwardly toward the nostril or outwardly toward the cheek (5 *A* and *B*). The inward procedure was recommended to reduce the outer periphery of the nasal attachment of the nasal wing without diminishing the size of the nostril or vestibulum itself. The outward procedure shortens the circumference and size of the nostril and vestibulum without transforming the outer periphery. The two kinds of triangular excision seem to be very similar. In both, *a* and *c* are the points which have to be moved to *b* and *d*, points which appear relatively more fixed. The only difference is their position in the outer or inner marginal lines. There is no difficulty in transferring point *a* to point *b* if both points are situated in the inner rim of the nostril.

6. Aufricht, G.: A Few Hints and Surgical Details in Rhinoplasty, Laryngoscope **53**:317-335 (May) 1943.

If they are located in the outer marginal line an additional incision (*i*) is required. For this purpose the ala is separated from the cheek in a line which is the continuation of incision *cd*. The length of this incision is directly proportional to the amount of shifting required and serves as a sliding guide for the approximation of the wound margins. The incision (*i*) for the act of displacement should follow the alar-facial fold in the skin in order to procure an external scar as inconspicuous as possible. The wedge-shaped section of tissue (*t*) is removed intranasally.

2. A rectangular, more or less parallelopiped-shaped piece of tissue with parallel sides of any required size from the full thickness of the ala (fig. 5 *C* and *D*). It may be recalled that an incision (*i*) is necessary to create room for the shifting and reattachment. The surgeon

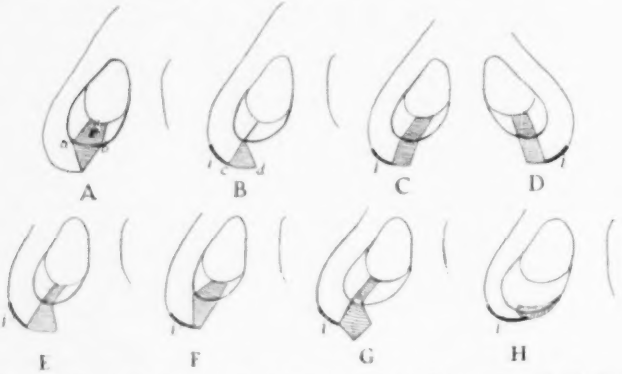

Fig. 5.—Standard surgical procedures; further details are given in the text. Sites of excisions are shaded.

can select the direction of the excision of the tissue either straight or toward the lateral side or the median line. In any case the procedure must be reversed in the left and in the right nostril. The result is a diminution of size and curvature of the nostril and of the outer circumference of the ala.

3. A trapezoidal excision with the broader side either at the inner or at the outer rim of the ala. Such an incision will result in the relative reduction of both margins, in a displacement of tissue at different levels (fig. 5 *E*, *F*). An additional incision (*i*) for shifting must be done later.

4. The reduction of the outer periphery can be augmented further by a pentagon-like incision with the fifth angle excising a wedge from the nasolabial ridge (fig. 5 *G*).

5. A segmental incision, useful at the base of the nostril, with an additional incision in the alar-facial groove (*i*) for shifting the alar wing (fig. 5 *H*).

RECONSTRUCTION OF THE ATROPHIC LOBULE

In a recent issue of the ARCHIVES OF OTOLARYNGOLOGY I [7] reported on a new intranasal technic employed in lengthening a short columella, reducing an excessively wide hypertrophic lobule and changing the direction of the nostrils. This technic consisted in the use of bent scissors directed around the crescent circumference of the septal cartilage toward the nasal spine. The prime purpose of this operative procedure was to protect the shifting mechanism of the upper lip.

Occasionally one sees a deformity of the base of the nose in which the portion between the nasal tip and the columella—i. e., the lobule—is atrophic. Figure 6 shows two such nasal bases before and after reconstruction.

The difficulties encountered in obtaining a satisfactory end result are common knowledge. But there is no particular method recorded in the available literature to date for correction of the atrophic lobule.

The following technical steps are employed. The procedure preliminary to all correction of the nares is the exposure of the lower lateral cartilages.

Infiltration anesthesia is performed with a 1 inch (2.5 cm.) 24 gage needle with a syringe containing a 1 per cent solution of procaine hydrochloride with 5 drops of 1:1,000 epinephrine hydrochloride solution to the ounce (30 cc.). The solution is injected subcutaneously about the external nasal orifice from inside each nostril at the tip, at the columella and along the alae.

With a Bard-Parker no. 11 knife an incision is made along the limen nasi—the projecting shelf of the lower margin of the upper lateral cartilages. The aponeurosis connecting the upper and lower lateral cartilages is incised, the slicing incision beginning at the center of the plica and extending ventrally (anteriorly) to the septum. The procedure is repeated on the opposite side. A button end knife is passed from one incision to the other, and the membranous septum is cut through, the knife being held close to the inferior margin of the septal cartilage.

A semicircular incision by means of a Bard-Parker knife no. 15 is now made within the nasal vestibule of each side just above the alar rim—that is, just below the inferior margin of the lower lateral catilage. A small scissors is then introduced into the incision between the skin and the cartilage. The blades are opened and the overlying tissues separated from the cartilage. The scissors are directed toward the initial intercartilaginous incision, separating the entire lateral crus of the lower lateral cartilage, and toward the septum mobile, separating the angle and the mesial crus of the lower lateral cartilage. With a dural hook placed at exactly the angle of junction between the lateral and mesial crura, the cartilage is drawn out of the vestibule and divided at the angle by means of a Bard-Parker no. 11 knife.

7. Griesman, B. L.: Suspension Mechanism of the Upper Lip and the Columella, Arch. Otolaryng. **50**:36-44 (July) 1949.

The definite location of the separation must be determined in each case. If a projecting tip is desired, a separation slightly lateral to the angle lengthens the lobule. If a normal-sized lobule and columella are present and only the dome of the tip has to be changed, a separation slightly medial to the angle will have to be chosen.

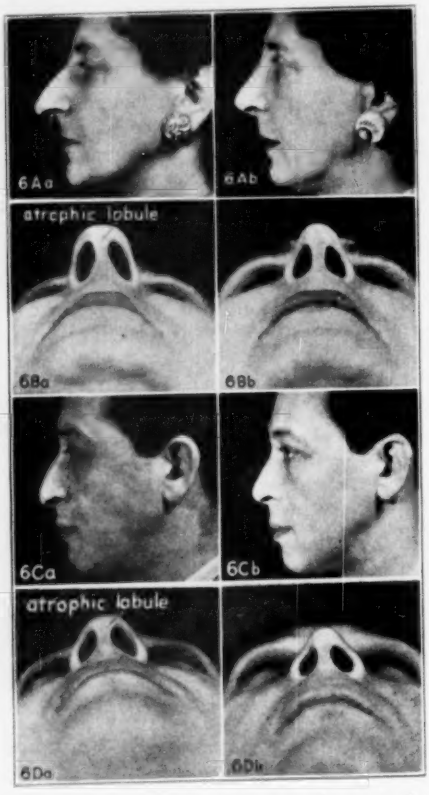

Fig. 6.—Atrophic lobules before and after correction.

The nasal bases shown in figure 6 *Ba* and *Da* have in common a wide tip, oversized nares and the cheeked type of alar-facial junction. The principal deformity is the atrophy of the lobule. The same operative steps were carried out in the two cases: the removal of a small nasal hump, narrowing of the nasal dorsum by osteotomy and shortening of the elongated nose by resection of a triangular piece of septal car-

tilage. The thickness of the base of the columella was reduced in both cases by excision of the excess of subcutaneous tissue and cartilage. In spite of the fact that this procedure theoretically seems to increase the size of the already oversized nares, the final end result (fig. 6 *Bb* and *Db*) proves the opposite. The voluminous elongated nostrils with the cheeked junction of the alar wings were corrected by excision of a quadrangular piece of tissue at the location of the alar-facial junction. The wound margins were coapted with atraumatic sutures.

In both cases a small strip of cartilage together with the still adherent vestibular skin was resected from the superior margin of the entire length of the lateral crura (fig. 7 *A*).

The operative steps used for the reconstruction of the lobule were a little different in the two cases because the patient shown in figure 6 *Ba* had an elongated columella combined with an atrophic lobule and a far projecting nasal tip, whereas the patient shown in figure 6 *Da* had a deficient lobule combined with a hanging nasal tip.

The extremely prominent pointed nose of the first patient (fig. 6 *Aa*) demanded the reduction of the excessively projecting septal cartilage by shaving off the anterior (ventral) and lower (caudal) margins. The columella was reattached to the reduced nasal septum by so-called orthopedic sutures. This technic consists of passing the sutures obliquely. When the stitches are tied, the columella will be brought backward (or forward). However, the effect cannot be achieved automatically by the stitches alone.

The main factor in getting good results with the orthopedic sutures in noses with a high degree of projection is the shoving back of the nasal tip during the tying of the stitches. In order to achieve this the surgeon uses his left hand to press the skin of the nasal dorsum downward against the septal cartilage and with the fingers of his right hand he pushes the columella as far down as possible against the anterior nasal spine, which was resected in most cases beforehand. Only in this way will the orthopedic suture attain the desired effect in reducing a very prominent nasal profile. Occasionally a submerged suture will conclude the resetting of the columella by marking a new subnasal point.

In the first patient (*6Aa*) 5 mm. of vestibular skin was then removed from the cut ends of the lateral crura (fig. 7 *B*). The vestibular skin was grasped with a very fine forceps, separated with a small scissors and resected. The deepithelized portions of the cartilage were then trimmed with fine scissors into an oval shape and brought together by means of a McKenty elevator over the mesial crura to be covered as with a roof (fig. 7 *C*). The forward extending base with its projecting tip was corrected by shortening and trimming the upper ends

of the mesial crura and by the deepithelization of the cut ends of the trimmed lateral crura forming an arch (Fomon).*

A large button prepared from the patient's own septal cartilage without perichondrium was then transplanted beneath the skin into the tip region just above the arch of the deepithelized cut ends of the lateral crura to increase the height of the lobule (fig. 7 *C*). Figure 6 *Bb* shows the end result: a straight columella, diminished circumference of the nares, and a reconstructed lobule.

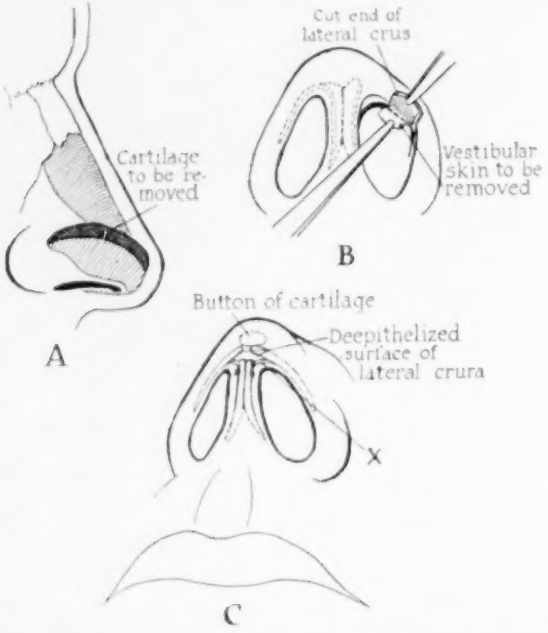

Fig. 7.—*A*, area of excision of redundant cartilage and vestibular skin (shaded); *B*, deepithelization of the cut end of the lateral crus; *C*, deepithelized lateral crura forming a roof over the mesial crura (sutured together). Point *x* indicates the location where the vestibular skin of the mesial and lateral crura grows together. A modeled button of cartilage has been inserted above the lateral crura to restore the contour of the lobule.

Figure 6 *Ca* shows the second patient before the rhinoplasty. The principal details are a hanging nasal tip, flaring alae, a cheeked junc-

8. Experience has shown that great care must be exercised in trimming the cut ends of the lateral crura of the lower lateral cartilages. After they have been replaced, they should point toward the nasal tip, not toward the dorsum of the nose. Fine atraumatic sutures at the upper lateral corner of the vestibule hold them in the right position and prevent them from falling backward.

tion, voluminous nares of unequal size and atrophy of the lobule. The height of the lobule is nearly equivalent to the thickness of the alar wings.

The problem of repair in reconstructing the lobule consisted in raising the nasal tip at the same time.

After the angle of the lower lateral cartilages was cut slightly laterally, 5 mm. of vestibular skin was then removed from the cut ends of the mesial crura in order to extend the nasal tip forward (fig. 8 *A*). The cut ends of the mesial crura were sutured together with very fine surgical gut sutures and trimmed into a rounded shape. The alar wings were taken in by resection of a parallelopiped-shaped piece of tissue.

A large button prepared from the resected hump was transplanted into the nasal tip to give the lobule its definite shape and to advance the tip to the desired level (fig. 8 *B*). A proper-sized lobule was constructed and a tip-raising operation done.

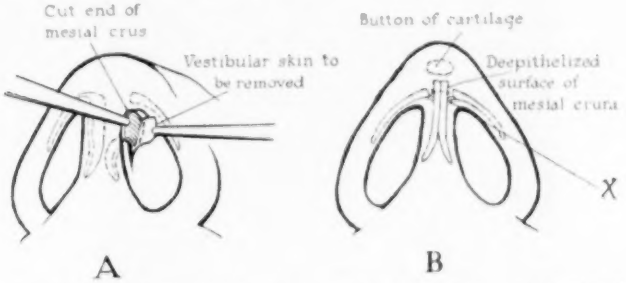

Fig. 8.—*A*, deepithelization of the cut ends of the mesial crura; *B*, deepithelized cut ends of the mesial crura sutured together and lengthened by insertion of a button of cartilage. Point *x* is the location at which the vestibular skin of the mesial and lateral crura grows together.

The difference in the operative steps in the two cases consisted in the location of the deepithelization of the cut ends, (1) of the lateral crura of the lower lateral cartilages (in case 6 *Aa*) and (2) of the mesial crura of the lower lateral cartilages (in case 6 *Ca*). The reason for the different methods will be easily understood. The vestibular skin of the mesial and the lateral crura grows together, as shown in figure 7 *C* and 8 *B* at *X*.

The use of a graft of a button of cartilage in the nasal tip accomplishes three things: 1. The tip is raised to a level about 2 mm. above the nasal dorsum. 2. The lobule is lengthened. 3. A cleft tip as end result, varying from a just visible dimpling of the lobule to a real groove, is avoided. Two narrow strips of adhesive tape are helpful in immobilizing the graft. One strip is placed just across the lobule below the graft and another over the nasal tip above the graft.

There is always a possibility that the button of cartilage will slip away when the skin of the nasal tip is undermined and freed from its underlying structures. Fomon solved this problem by employing a pin batten-shaped cartilage as a graft. Here the button is secure because it is fixed to a strut and cannot move away or get lost.

Such a pin batten cartilage, with a head shaped like a button and a needle-like shaft, can be successfully used to buttress the lobule. For this purpose the graft is introduced into the soft tissues of the columella from the marginal incision along the anterior (caudal) border of the mesial crura which was previously made for the delivery of the lower lateral cartilages. The graft is then inserted just beneath the skin and caudal to the medial crura, extending from the nasal tip to the beginning of the inferior third of the columella. A mattress suture through the uppermost part of the columella fixes the pin batten cartilage in position.

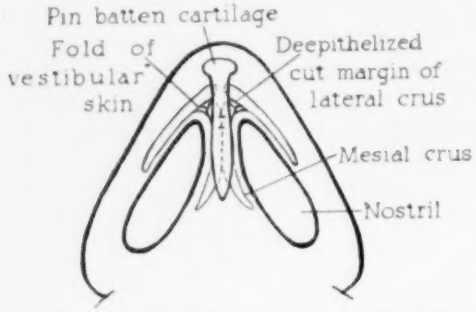

Fig. 9.—Pin batten cartilage grafted in front of the mesial crura. The head of the pin batten extends into the lobule; the shaft into the columella.

Figure 9 demonstrates the position of the pin batten-shaped cartilage, the head extending into the lobule and its longitudinal axis lying just in front of the mesial crura.

CORRECTION OF THE COLUMELLAR ALAR-FACIAL JUNCTION

It is generally agreed that no feature of the face can be considered to conform to absolute esthetic standards. Figure 10 *A* presents a profile with many elements of physical disfigurement, such as a long nose, displeasingly ill shaped owing to a prominent hump, a broad nasal tip, a wide lobule and a wide base of the columella. After rhinoplasty—that is, removal of the hump, moderate shortening of the length of the nose, narrowing of the base of the columella by subcutaneous excision of connective tissue, lateral osteotomy and deepithelization of the cut ends of the lateral crura of the lower lateral cartilages—there was evidence that the new structure of the nose did not conform with the

previous columellar type of alar-facial junction. The nostrils appeared distended. At the conclusion of the rhinoplasty it was felt necessary to alter the shape of the columellar attachment into the form of a labial attachment.

The surgical problem consisted in changing the line of the inner rim of the alae, leaving the outer rim of the alae undisturbed, as shown in figure 5 *A*. There was no need to shift the ala foot as a whole. As can be seen in figure 10 *D*, no incision was made for displacement of the foot. A triangular piece of tissue with its apex directed outwardly toward the cheek was excised (shown at *x* and *y* in figure 10 *C*). Figure 10 *D* shows the end result. The previous columellar alar-

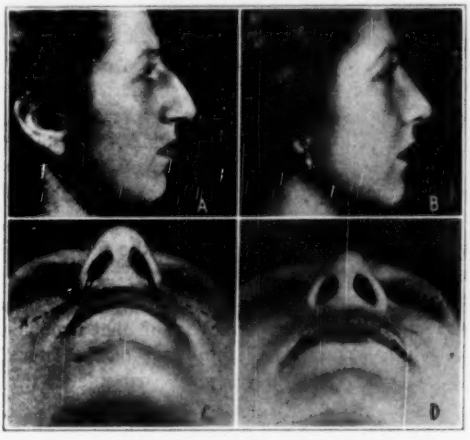

Fig. 10.—*A*, profile before rhinoplasty; note the mildly retracted columella and rounded nasal tip. *B*, profile after rhinoplasty. *C*, nasal base of the same patient before rhinoplasty; note the broad lobule, wide base of the columella, enlarged nostrils and columellar type of alar-facial junction. *D*, same base after plastic repair; note the changed nasal tip and columella, narrowed nostrils and labial type of alar-facial junction. The letters *x* and *y* indicate the sites of excisions of small triangular pieces of tissue with the base extending into the nares and the apex directed toward the cheek; another geometric form of excision was not indicated because of the lack of excessive outer curvature of the alae.

facial junction is replaced by a labial alar-facial junction. The formerly blunt nasal tip and the wide lobule have become transformed into a mildly pointed tip and small lobule.

SUMMARY AND CONCLUSIONS

1. The base of the nose shows a rather complex construction. Eight structural members are described: the tip, the lobule, the columella, the alae, the alar-facial junction, the nasolabial ridge, the nos-

trils and the subnasal point. The length of the base is made up by the height of the lobule plus the height of the columella.

2. The individual variations of the alar facial junction can be classified into three types. I suggest the terms (1) cheeked, (2) labial and (3) columellar. The difference comes from the attachment of the foot of the alar shaft. When the foot appears absorbed into the cheek, the junction should be termed "cheeked"; when it is joined to the upper lip, the junction is termed "labial." When the foot is extended into a tubule several millimeters in length and stretched along the inferior border of the nostril or taking an upward course along the columella, the junction is called "columellar."

3. The standard surgical procedures are classified into two main types: (1) the "marginal" incisions and (2) the "encircling" incisions. The marginal incisions extend into the margin of the nostril and can be performed with or without resection of tissue from the alar wing or the alar-facial junction. Five standard surgical procedures are recommended as marginal incisions to correct deformities of the alae. The excision of a triangular, rectangular, trapezoidal, pentagon-like or segmental-shaped piece of tissue corrects the size and direction of the nostrils and the contour of the alae by changing the outer or inner border of the alar wings.

4. The Fomon method of denudation of the cut ends of the lateral crura of the lower lateral cartilages has proved to be a satisfactory improvement in modeling the lobule.

5. Two operations for reconstruction of the atrophic lobule are described. Deepithelization of the cut ends of the lateral or mesial crura of the lower lateral cartilages can be considered the method of choice.

6. The grafting of a button of cartilage (autogenous or preserved) beneath the freed skin of the tip accomplishes three purposes: (1) raising the tip, (2) lengthening the lobule and (3) preventing a cleft nasal tip.

7. The problem of fixation of the button was solved by the pin batten graft (Fomon). A strut of cartilage with a head and a pointed shaft is transplanted in front of the mesial crura just beneath the skin. The head of the pin elevates the nasal tip over the dorsum and at the same time lengthens the lobule.

3 East Sixty-Sixth Street.

ENDOLYMPHATIC HYDROPS WITHOUT VERTIGO

Its Differential Diagnosis and Treatment

HENRY L. WILLIAMS, M.D.

BAYARD T. HORTON, M.D.

AND

LOIS A. DAY, M.D.

ROCHESTER, MINN.

THE CARDINAL symptom of Ménière's[1] disease is usually stated to be vertigo. The cardinal sign, however, appears to be inner ear deafness involving the low tones rather than the high, and more apparent on the bone conduction curve determined with the audiometer than on the air conduction curve.

Use of the term "Ménière's disease" fixes the mind of the observer on the triad of symptoms originally described (vertigo, nausea and deafness) and has prevented attention being given to the possibility that in this condition loss of hearing might occur without development of vertigo and nausea. Since the observed pathologic change appears to have its initial expression in the scala media and the symptom of vertigo is probably produced by a fortuitous deformity of the utricle, it seems possible that Ménière's disease has as its primary expression, and possibly may have as its only clinical symptom, loss of hearing. Lindsay[2] has described a case with the typical pathologic features of endolymphatic hydrops in which no history of vertigo or even of dizziness was obtained. That Ménière's disease affects the

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From the Section on Otolaryngology and Rhinology (Dr. Williams), the Division of Medicine (Dr. Horton) and the Section on Gynecology and Obstetrics (Dr. Day), Mayo Clinic.

1. Ménière, M. P.: Mémoire sur des lésions de l'oreille interne donnant lieu à des symptômes de congestion cérébrale apoplectiforme, Gaz. méd. de Paris **16**: 597-601, 1861.

2. Lindsay, J. R.: Labyrinthine Dropsy, Laryngoscope **56**:325-341 (July) 1946.

hearing has been pointed out by many authors. Mygind and Dederding [3a] described the fluctuating character of the loss of hearing in Ménière's disease and stated that by their method of treatment the hearing was improved in 81 per cent of their cases in which treatment was continued for three years or more. Mygind [3b] drew attention to the fact that although bone conduction of sound is decreased in Ménière's disease, the deafness is not necessarily a nerve type. He pointed out that the perilymph and the endolymph are a part of the conducting mechanism of the ear. He stated that pressure fixing the windows, whether exerted from the middle ear or, as in Ménière's disease, resulting from increased pressure of the endolymph, would produce a decrease in bone conduction and interfere more with the transmission of low tones than with that of high. He suggested that the deafness in Ménière's disease be classified as primarily an inner ear deafness of the conduction type. He pointed out that nerve deafness might occur secondarily in Ménière's disease from the effect of continuous increased pressure of the endolymph. In such a case inner ear deafness made up of a combination of conductive and nerve elements would be present. That part of the deafness produced by the conditions interfering with the conducting mechanism of the inner ear might be reversible, whereas that part produced by degeneration of the neural elements of the organ of Corti would be irreversible. Crowe [4] stated that the fluctuating character of the deafness was good ground for suspecting that the cause of Ménière's disease was increased fluid pressure in the labyrinth; this suggestion anticipated the histologic observations of Hallpike and Cairns.[5] Lillie, Horton and Thornell [6] were able to demonstrate that intravenous administration of histamine phosphate could improve the hearing of certain patients having Ménière's disease.

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3. (*a*) Mygind, S. H., and Dederding, D.: IV. The Pathogenesis of Ménière's Disease and of Kindred Conditions in the Ear and the Rest of the Body, Ann. Otol., Rhin. & Laryng. **47**:938-946 (Dec.) 1938. (*b*) Mygind, S. H.: Experimental-Histological Studies on Labyrinth: Correlation of Our Histological and Clinical Experiments and Observations; Conclusive Review, Acta oto-laryng. **35**: 108-129, 1947.

4. Crowe, S. J.: Ménière's Disease: A Study Based on Examinations Made Before and After an Intracranial Division of Vestibular Nerve, Medicine **17**: 1-36 (Feb.) 1938.

5. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, Proc. Roy. Soc. Med. **31**:1317-1336 (Sept.) 1938; J. Laryng. & Otol. **53**:625-654 (Oct.) 1938.

6. Lillie, H. I.; Horton, B. T., and Thornell, W. C.: Ménière's Symptom Complex: Observations on the Hearing of Patients Treated with Histamine, Ann. Otol., Rhin. & Laryng. **53**:717-741 (Dec.) 1944.

Harris and Moore [7] also noted improvement in hearing on administration of nicotine acid in their cases of Ménière's disease.

It seemed a reasonable assumption, therefore, that patients might be found who have a type of deafness characteristic of Ménière's disease but who do not give a history of vertigo and whose hearing can be improved by appropriate treatment.

THE PATHOLOGIC ASPECTS OF MÉNIÈRE'S DISEASE AND A SUGGESTED NEW DESCRIPTIVE NAME

The discovery by Hallpike and Cairns that the true pathologic picture of so-called Ménière's disease is a gross dilation of the endolymphatic or membranous labyrinth which affects chiefly the scala media with secondary dilation of the saccule and utricle has disposed of much of the controversy in regard to the causation of the condition. These pathologic observations of Hallpike and Cairns and Hallpike and Wright [8] have subsequently been confirmed by Rollin,[9] Yamakawa,[10] Altmann and Fowler Jr.[11] and Lindsay.[12] Since so many competent observers in different countries have found the same pathologic picture in authenticated cases of Ménière's disease, there can now be little doubt that, as Crowe pointed out previously, the condition should be known as a disease since it has an invariable pathologic background and a regular and characteristic clinical expression.

Lindsay [12] suggested that the condition be called "labyrinthine dropsy" but stated that in the hydrops of Ménière's disease the disturbance is confined to the endolymphatic system and is not in the perilymph. In contrast, the labyrinthine reaction which occurs in serous labyrinthitis affects the perilymphatic spaces primarily, while changes in the endolymphatic system may be limited to dilation and degenerative changes of

7. Harris, H. E., and Moore, P. M., Jr.: The Use of Nicotinic Acid and Thiamin Chloride in the Treatment of Ménière's Syndrome, M. Clin. North America **24**:533-542 (March) 1940.

8. Hallpike, C. S., and Wright, A. J.: On the Histological Changes in the Temporal Bones of a Case of Ménière's Disease, J. Laryng. & Oto. **55**:59-65 (Jan.) 1940.

9. Rollin, H.: Zur Kenntnis des Labyrinthhydrops und des durch ihn bedingten Ménière, Hals-, Nasen- u. Ohrenh. **31**:73-109 (March) 1940.

10. Yamakawa, K.: Ueber die pathologische Veränderung bei einen Ménière-Kranken, Ztschr. f. Laryng., Rhin., Otol. **34**:181, 1938.

11. Altmann, F., and Fowler, E. P., Jr.: Histological Findings in Ménière's Symptom Complex, Ann. Otol., Rhin. & Laryng. **52**:52-80 (March) 1943.

12. Lindsay, J. R.: Labyrinthine Dropsy and Ménière's Disease, Arch. Otolaryng. **35**:853-867 (June) 1942.

the sense organs and the stria vascularis. It appears, therefore, that the term "endolymphatic hydrops" is more descriptive than "labyrinthine dropsy" in that it excludes inflammatory conditions, such as serous labyrinthitis, from consideration. In other words, endolymphatic hydrops is an extracellular edema involving primarily the scala media of the cochlea.

SYMPTOMS OTHER THAN DEAFNESS ASSOCIATED WITH ENDOLYMPHATIC HYDROPS

Müller [13a] and Mygind and Dederding, in examining patients with the triad vertigo, deafness and tinnitus, found that these patients often had severe unilateral headaches, angioneurotic edema (Quincke's edema), dermatographia, hay fever and other conditions known as allergic, that they were of the neurotic type, fatiguing easily, and that they had low blood pressure. In examining the capillary systems of persons exhibiting such symptoms without concomitant endolymphatic hydrops, Müller found that they also had as the pathologicophysiologic basis of their symptoms a so-called spastic-atonic condition in certain capillaries. The findings of Müller in regard to the incoordination between the arterioles and the capillary loop have been confirmed by the investigations of Brown.[13b]

After Müller, Mygind and Dederding and others pointed out that many patients with vertiginous endolymphatic hydrops had many other symptoms and signs to indicate that they were of the vagotonic type, it was decided that young persons who had perceptive deafness and gave clinical evidence of such so-called allergic (or pathergic) conditions as hay fever, vasomotor rhinitis, urticaria, palindromic rheumatism and the like might have endolymphatic hydrops without vertigo. Also, it was observed that the symptom triad of Ménière tended to develop in certain cases during and after the menopause. It seemed likely, therefore, that women who exhibited the first indication that deafness was developing during the menopause or who exhibited increased deafness during menstrual periods might have endolymphatic hydrops. On a theoretic basis, in certain cases sudden deafness might be either on the basis of endolymphatic hydrops or on that of thrombosis of the internal auditory artery or of its cochlear branch. It was found that for persons who had unilateral loss of hearing or marked differences in the degree of deafness in the two ears, testing for binaural diplacusis as described by Shambaugh and

13. (*a*) Müller, O.: Die feinsten Blutgefässe des Menschen in gesunden und kranken Tagen, ed. 2, Stuttgart, Ferdinand Enke, 1939. (*b*) Brown, G. E.: The Skin Capillaries in Raynaud's Disease, Arch. Int. Med. **35**:56-73 (Jan.) 1925.

Roberts [14] was of considerable help in coming to a conclusion as to the type of deafness present. In those who had diplacusis, an impending improvement of the hearing was often forecast by the disappearance of diplacusis.

The criteria finally established for a tentative diagnosis of endolymphatic hydrops, which were used also in selecting patients for treatment in our series, were as follows: 1. Patients who had inner ear deafness with a tendency toward greater loss of hearing for low tones than for high tones were considered to have this condition. This tendency often was more apparent on the bone conduction curve than on the air conduction curve of the audiogram. 2. A history of considerable variability of the degree of deafness was another criterion. In women increased deafness before, during and shortly after the menstrual period was considered significant. 3. A history of vasomotor rhinitis, urticaria, dermatitis factitia or severe unilateral headache, often associated with increased deafness, was felt to suggest that the deafness might be due to endolymphatic hydrops. 4. A sudden decrease of ability to hear, occurring in a period of two to three days, followed by persistence of the hearing loss was thought to indicate the possibility that the disorder was due to endolymphatic hydrops.

THE THEORETIC BASIS OF THE SELECTION OF TREATMENT IN CASES OF ENDOLYMPHATIC HYDROPS

Müller described an inherited tendency in certain persons to react in adult life to certain preferred stimuli, usually physical in nature, by the formation of an abnormal capillary picture. In these persons, in certain localized regions the smooth muscle collar at the beginning of the capillary loop contracted tetanically. This spasm produced anoxia in the tissue cells nourished by the capillary loop; the capillary epithelium then became atonic; the permeability of the capillary wall was increased, and the fluid and electrolyte metabolism were disturbed in the affected area. Lewis [15] showed that under ischemic conditions an "H substance," which he thought might be histamine, was released from the affected tissues. Horton, Brown and Roth [16]

14. Shambaugh, G. E., Jr., and Roberts, W. S.: Significance of Diplacusis in Ménière's Syndrome, in Medical Papers Dedicated to Henry Asbury Christian, Physician and Teacher, in Honor of His Sixtieth Birthday, Baltimore, Waverly Press, Inc., 1936, pp. 894-902.

15. Lewis, T.: Suggestions Relating to the Study of Somatic Pain, Brit. M. J. **1**:321-325 (Feb. 12) 1938.

16. Horton, B. T.; Brown, G. E., and Roth, G. M.: Hypersensitiveness to Gold, with Local and Systemic Manifestations of a Histamine-Like Character: Its Amenability to Treatment, J. A. M. A. **107**:1263-1269 (Oct. 17) 1936.

later submitted evidence that the substance released was actually histamine. Lewis also produced evidence that the skin could be sensitized to physical agents and that such sensitization was associated with release of a chemical substance, probably histamine, on appropriate stimulation.

Selye[17] demonstrated that on being exposed to physical and emotional stimuli, which he termed "alarming" stimuli, animals reacted with signs of shock in the first stage (the "alarm reaction") of the general adaptation syndrome. As is well known, the signs of shock are associated with an increase of the histamine in the blood as has been demonstrated in urticaria by Rose.[18] Selye found that in the shock phase of the alarm reaction the potassium content of the blood rises as Talbott and Brown[19] observed it to rise during the attack phase of vertiginous endolymphatic hydrops. Selye also pointed out evidence that the mechanism of adaptation plays a part in the release of antibodies in serologic immune reactions, a defect of which is thought to play a part in antigen-antibody allergy. Selye also mentioned the part played by the endocrine system in the general adaptation syndrome.

Duke[20] observed in certain persons an abnormal reaction elicited by physical and emotional stimuli. This physical allergy was of two types: (1) the contact type and (2) the reflex type in which a stimulus acting on a body surface produced an "allergic" reaction in a distant area through the mediation of the autonomic nervous system. Duke suggested that the mechanism involved in physical allergy differed from that of the ordinary antigen-antibody type. Release of histamine has been demonstrated in cases of allergy by Katz.[21] In antigen-antibody allergy this release is thought to be due to the junction of the antigen and an antibody still fixed to a cell, with cellular injury resulting. In physical allergy the mechanism is thought to be the spastic-atonic state of the capillaries described by Müller. The hypotheses of Müller, Lewis, Selye, Duke and others dovetail so perfectly that they form a mutually supporting arch for the hypothesis that so-called physical allergy is associated with the presence of histamine released

17. Selye, H.: The General Adaptation Syndrome and the Diseases of Adaptation, J. Clin. Endocrinol. **6**:117-230 (Feb.) 1946.

18. Rose, B.: Studies on Blood Histamine in Patients with Allergy: II. Alterations in the Blood Histamine in Patients with Allergic Disease, J. Clin. Investigation **20**:419-428 (July) 1941.

19. Talbott, J. H., and Brown, M. R.: Ménière's Syndrome: Acid-Base Constituents of the Blood; Treatment with Potassium Chloride, J. A. M. A. **114**: 125-130 (Jan. 13) 1940.

20. Duke, W. W.: Allergy: Asthma, Hay Fever, Urticaria and Allied Manifestations of Reaction, St. Louis, C. V. Mosby Company, 1925.

21. Katz, G.: Histamine Release in the Allergic Skin Reactions, Proc. Soc. Exper. Biol. & Med. **49**:272-277 (Feb.) 1942.

by stimuli transmitted by the autonomic nervous system and that the release of histamine is localized to the regions of abnormal capillary activity. That endolymphatic hydrops is a form of physical allergy probably involving the capillary loops of the stria vascularis is supported by the pathologic changes described. It is improbable that the picture of localized extracellular edema can be produced in the body by any other agency than allergy (pathergy).

The effect of the endocrine glands on the release of histamine and its effects has been mentioned by many authors, including Müller and Selye. Farmer and Fribourg [22] noted that the feeding of thyroid extract to guinea pigs enhances their susceptibility to histamine and their anaphylactic response. Farmer [23] also found, as did Selye, that the sensitivity of a species to histamine and subsequently its anaphylactic response are determined by its adrenocortical hormone. Selye also stated that increase in permeability of capillary walls is present in the shock phase of the alarm reaction. If Müller's hypothesis that the fundamental cause of endolymphatic hydrops is a spastic-atonic condition of the capillaries of the stria vascularis is correct, the relief obtained by vasodilators might depend on their ability to release the spasm of the lock muscle of the arteriole at the beginning of the affected capillary loop, and to produce other effects. These effects consist of sweeping the dilated and varicose portion free of any histamine, supplying oxygen and eliminating the effects of anoxia on the epithelium of the capillary wall and on the leukocytes caught by the stasis of the capillary blood stream. If this hypothesis is correct, any vasodilator ought to produce comparable relief in Ménière's disease. The relief of the symptoms of endolymphatic hydrops with vertigo (Ménière's disease) produced by vasodilators as far apart chemically as papaverine, histamine, neostigmine, nicotinic acid and solutions of magnesium sulfate has substantiated this hypothesis.

Since Horton had found that the highest percentage of improvement in patients having the triad of symptoms described by Ménière was obtained by daily intravenous administration of a solution of histamine phosphate, it was decided to try this method of treatment on certain patients presenting the findings of inner ear deafness whose deafness might depend on endolymphatic hydrops without vertigo rather than on degeneration of the cells of the organ of Corti, their neuraxons or their central connections.

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22. Farmer, L., and Fribourg, R.: Studies on Histamine Sensitivity and Anaphylactic Response: The Effect of Thyroid Extract, Proc. Soc. Exper. Biol. & Med. **50**:208-209 (June) 1942.

23. Farmer, L.: Histamine Sensitivity and Anaphylactic Response: III. Theoretical Considerations, Am. J. Clin. Path. **13**:365-367 (July) 1943.

It was not until one of us (B. T. H.) in 1945 discovered that evidences of improvement in hearing often did not manifest themselves until after two weeks of daily intravenous treatment with histamine that we were able to achieve any measure of worth while results in the ordinary case.

THE EFFECT OF PREMENSTRUAL TENSION ON DEAFNESS DUE TO ENDOLYMPHATIC HYDROPS

It was found that in certain women who gave evidence at their menstrual periods or during or following the menopause that certain disturbances of the endocrine glands were present, concomitant treatment with histamine and estrogens was necessary to secure a favorable result in relieving loss of hearing.

There is evidence that some relationship exists between the genital function of women and water metabolism. It is generally accepted that a significant increase of weight and occasional edema may occur in some women at the menses. Okey and co-workers [24] noted a premenstrual gain in weight in their patients who had menstrual disorders and premenstrual headaches. Thorn and his co-workers [25] observed the weight of 50 normal women aged 19 to 30 years each day for two months. They found that 24 gained 1 Kg. or more before menstruation and 76 per cent had a similar gain at the time of ovulation. About two thirds of those who gained weight before menstruation also had swelling of the abdomen and increased appetite and thirst at that time.

Studies to elucidate the etiologic factors concerned in this gain of weight were carried out on dogs. In short experiments they found that estradiol, estrone, progesterone, pregnanediol and testosterone cause retention of sodium and chlorides and simultaneous reduction of the volume of urine and increase of body weight. This was followed, after five days, by a rebound effect during which the retained sodium, chlorides and water were excreted.

In women Thorn and his co-workers noted that sodium, chlorides and water were retained at the time of ovulation and a few days before

24. (*a*) Okey, R., and Boyden, R. E.: Studies of the Metabolism of Women: III. Variations in the Lipid Content of Blood in Relations to the Menstrual Cycle, J. Biol. Chem. **72**:261-281 (March) 1927. (*b*) Okey, R., and Stewart, O.: Diet and Blood Cholesterol in Normal Women, ibid. **90**:717-727 (Feb.) 1933.

25. (*a*) Thorn, G. W., and Harrop, G. A.: "Sodium Retaining Effect" of Sex Hormones, Science **86**:40-41 (July 9) 1937. (*b*) Thorn, G. W.: Nelson, K. R., and Thorn, D. W.: A Study of the Mechanism of Edema Associated with Menstruation, Endocrinology **22**:155-163 (Feb.) 1938. (*c*) Thorn, G. W., and Engel, L. L.: Effect of Sex Hormones on Renal Excretion of Electrolytes, J. Exper. Med. **68**:299-312 (Sept.) 1938. (*d*) Thorn, G. W., and Emerson, K., Jr.: The Role of Gonadal and Adrenal Cortical Hormones in the Production of Edema, Ann. Int. Med. **14**:757-769 (Nov.) 1940.

the menses. In 2 subjects studied at the time of ovulation the output of estrogen in the urine was increased. In concluding a paper, Thorn and his associates said, "The increase in the secretion of the water and salt retaining sex hormones and the increase in appetite and thirst appeared to be contributing factors in the body weight gain which occurs during the menstrual cycle." In this paper the authors seem to feel that estrogen is the hormone most likely to be responsible for this effect. In a later study they said, however, that the female sex hormone which plays the most important role in regulating electrolyte balance cannot be stated definitely. "Certainly the retention of electrolytes during the premenstrual period is much more striking than that which is observed at the time of ovulation." They pointed out that this suggests the possibility that progesterone or progesterone added to estrogen is more concerned with retention of electrolytes and water than estrogen alone. Their experimental studies demonstrated that the effect of progesterone on electrolyte balance more closely resembles the effect of the adrenal cortical hormone than that of either estrone or testosterone. They said further that the salt and water retaining effects of the sex hormones may possibly be mediated in part or entirely through other glands of internal secretion.

Teel and Reid [26] found that in rats excretion of water was not inhibited by the injection of estrone and estradiol in short experiments. They suggested that the retention of water might be due to estrogenic stimulation of the pituitary gland.

Robinson and Farr [27] reported finding moderate amounts of an antidiuretic substance in the urine of a patient with recurring menstrual edema during the period when edema was present. None was found in the urine during the edema-free interval.

A complete understanding of the exact nature of the physiologic disturbance in this condition awaits elucidation not only of the role played by the various sex hormones but also of the complex part that the liver plays in the regulation of body water.[28]

Clinical observations of patients with the syndrome of premenstrual edema [29] has led one of us (L. A. D.) to believe that it develops in

26. Teel, H. M., and Reid, D. E.: Observations upon the Occurrence of an Antidiuretic Substance in the Urine of Patients with Pre-Eclampsia and Eclampsia, Endocrinology **24**:297-310 (March) 1939.

27. Robinson, F. H., Jr., and Farr, L. E.: The Relation Between Clinical Edema and the Excretion of an Antidiuretic Substance in the Urine, Ann. Int. Med. **14**:42-54 (July) 1940.

28. Labby, D. H., and Hoagland, C. L.: Water Storage and Movements of Body Fluids and Chlorides During Acute Liver Disease, J. Clin. Investigation **26**:343-353 (March) 1947.

29. Day, L. A.: Premenstrual Edema, S. Clin. North America **27**:827-836 (Aug.) 1947.

women with varying degrees of ovarian dysfunction. The diagnosis of ovarian dysfunction is made by study of the patient's menstrual and reproductive histories. Laboratory tests of confirmatory value are the determination of the basal metabolic rate, assays of the urine for the amounts of excreted estrogen, chorionic gonadotropin and pregnanediol glucuronidate and a histologic study of the endometrium in relation to the menstrual cycle. Local pelvic lesions should be ruled out by careful physical examination.

Therapy directed to improving the ovarian function combined with administration of agents used in combating retention of water has usually resulted in some improvement in the patient's condition. Ovarian function may be improved by the use of a high protein, high vitamin diet and by following habits of good hygienic living. Endocrine therapy consists of the use of thyroid to elevate a low basal metabolic rate and the administration of a small dose of estrogen. Adherence to a diet low in salt and the administration of a diuretic, such as potassium nitrate, are helpful in preventing retention of water.

We have found that in certain women premenstrual edema reenforced the symptoms produced by endolymphatic hydrops.

THE POSSIBLE INFLUENCE OF PSYCHOGENIC DEAFNESS ON THE RESULTS OBTAINED

We have tried to exclude the possible influence of psychogenic deafness on our results by keeping in mind the differential diagnosis between this type and other types of perceptive deafness.

Semenov [30] stated that the most significant symptoms and signs which indicate that a patient's deafness is psychogenic are (1) an obscure or a sudden onset of deafness; (2) complete absence of hearing in both ears; (3) total absence of hearing in one ear and severe deafness in the other; (4) flat audiograms; (5) variation in audiograms; (6) disproportion between the results of hearing tests; (7) normal vestibular response; (8) binaural deafness with pathologic change in one ear only; (9) severe shock or emotional distress prior to onset, and (10) a sudden improvement in hearing. We kept these points in mind while treating our patients and although we cannot state categorically that psychogenic factors have been absolutely eliminated, we feel that in none of the recoveries has psychogenic deafness been a factor.

For this study we reviewed the cases of perceptive deafness without vertigo observed at the Mayo Clinic, in the years 1945 and 1946. In those two years approximately 1,394 patients were seen whose condition was diagnosed as nerve (perceptive or inner ear) deafness. Among

30. Semenov, H.: Deafness of Psychic Origin and Its Response to Narcosynthesis, unpublished data.

these we treated 32 who we thought sufficiently met the criteria for the establishment of the diagnosis of endolymphatic hydrops without vertigo to warrant a trial of treatment. Twenty-two additional patients met these criteria but were not treated. It is apparent, therefore, that the condition of endolymphatic hydrops without vertigo is relatively uncommon, since it was encountered in less than 50 of 1,000 cases in which inner ear deafness was present. In this same period of two years there were 585 cases in which the diagnosis of Ménière's symptom complex was made. The great majority of these cases appeared to be instances of endolymphatic hydrops, so that attacks of vertigo appeared to be the rule rather than the exception in this malady.

As a control, 25 patients who were considered as having perceptive or nerve deafness and who had loss of hearing for high tones and preservation of hearing for low tones and otherwise did not meet the criteria for diagnosis of endolymphatic hydrops were treated by intravenous injection of histamine phosphate solution without improvement.

METHOD OF TREATMENT

The method is the same as that which one of us (B. T. H.) introduced in 1939 and has been followed since that time. The procedure is as follows: 2.75 mg. of histamine phosphate [31] diluted in 250 cc. of isotonic solution of sodium chloride is administered by the drip method at rates usually ranging from 16 to 60 drops per minute, depending on the tolerance of the patient. It is the rate of administration of the solution and not primarily the amount of histamine administered which is of paramount importance. This procedure is usually repeated daily. It is important for the patient to have food in his stomach at the time the histamine is given so that the excess gastric acids provoked by histamine can be readily absorbed. The rate of administration should be such that the patient has no headache and no discomfort whatsoever. The rate is gradually increased during the first treatment, and also during subsequent treatments, but is always kept below the reaction level. At no time is treatment continued for more than one and a half hours, and the patient never receives more than 250 cc. at each injection.

Reactions do not occur when the solution is given properly. Disposable tubing, needles and syringes which are free of pyrogens should be employed.

For women who had a severe decrease in their ability to hear just before and during the menses or at the time of ovulation, the giving of 0.1 mg. of diethylstilbestrol daily by mouth had a tendency to prevent this decrease in ability to hear. Diethylstilbestrol alone would not produce or maintain improvement in hearing, and histamine alone could not maintain the gain in hearing during either the premenstrual or the midmenstrual periods. With the two in conjunction, however, this tendency to lose hearing was controlled. Oftentimes the benefit was not apparent in the first month of treatment with diethystilbestrol but became apparent in the second month. If a low basal metabolic rate is found, the patient may be given sufficient desiccated thyroid to elevate the basal metabolic rate to the neighborhood of — 5 per cent.

31. Histamine diphosphate—Abbott is used.

After the maximal therapeutic result had apparently been secured, the patients were instructed in the subcutaneous administration of either histamine phosphate solution or monoethanolamine nicotinate solution ("nicamin") as maintenance therapy. From 0.10 to 0.30 cc. of a solution of 0.275 mg. of histamine phosphate was given daily for a month and then was given three times a week for another month. If symptoms, such as tinnitus, tended to return, the patients were instructed to administer the histamine solution subcutaneously until the symptoms again disappeared.

If "nicamin" was used, 1 cc. (conaining 25 mg. of monoethanolamine nicotinate) was injected subcutaneously. This dose was increased by a like amount daily until the patient was receiving 4 cc. of "nicamin" (100 mg. of monoethanol nicotinate). This dose was used for a month, and then the treatment was stopped. The patients were instructed to begin the injections again if any symptom, usually tinnitus, was noted.

Summary of Results in Thirty-Two Cases

	Total Number	Number in Which Results Were		
		Good	Fair	Nonbeneficial
Patients treated	32	11	8	13
Patients who had sudden onset of deafness	8	3	0	5
Patients whose deafness had been present:				
1 year or less	11	4	1	6
6 months or less	7	3	0	4

RESULTS OF TREATMENT

Thirty-two patients who met the criteria established for the diagnosis of endolymphatic hydrops without vertigo were treated.

A good result was considered to be a restoration of the hearing to conversational levels or better and abolition of tinnitus. All patients who had a worth while clinical result secured early abolition of tinnitus. A fair result was considered to be an improvement of hearing of 15 decibels or more for all the speech frequencies without restoration of hearing to conversational levels.

Of the 32 patients selected for treatment, 8 had unilateral and the rest bilateral loss of hearing. The results of treatment are shown in the table.

Patients whose deafness had begun suddenly appeared to have a fair chance of improvement, and the length of time the deafness had been present seemed to have some effect on the end result (table). Three patients were treated who had lost hearing in one ear several years previously and in the second ear six months or less before treatment. Hearing was restored to conversational levels in the

recently affected ear in all these patients, but little or no improvement occurred in the other ear. This seems to indicate that irreversible changes may take place in the nerve cells of the organ of Corti if it is subjected to the increased pressure of endolymph for a sufficiently long time.

In only 1 patient of the control series was there any suggestion that even the slightest improvement took place. In this patient an improvement of 10 decibels was noted in all frequencies. We believe

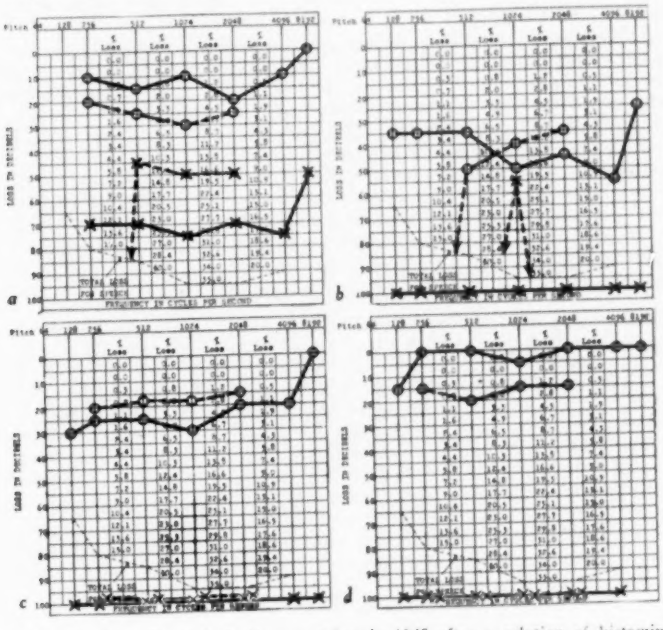

Fig. 1 (case 1).—(*a*) Audiogram taken in 1945 after a solution of histamine phosphate had been injected daily for one week; no improvement of hearing is shown. (*b*) Audiogram made in the following year, in August 1946. (*c*) Audiogram made after twenty-six daily intravenous injections of solutions of histamine phosphate (Aug. 5 to Aug. 31, 1946). (*d*) Audiogram made in September 1946, showing approximately normal hearing in the right ear. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

this improvement was apparent rather than real and was due to the errors inherent in audiometric testing.

ILLUSTRATIVE CASES

CASE 1.—A boy aged 14 came to the clinic in 1945 because of attacks of vertigo, nausea, vomiting and tinnitus associated with loss of hearing in the left ear. Symptoms had been present for two weeks. He also gave a history of having had

migraine. From six intravenous injections of histamine phosphate he derived relief from vertigo; no attempt was made to improve the hearing in the left ear, although this was noted to have improved slightly (chart 1*a*).

This patient returned in 1946. He stated that he had entered the Navy in 1945. In June 1945 he noticed that tinnitus, which had previously been present only in the left ear, was present in both ears. He now was found to have total loss of hearing in the left ear. The hearing of the right ear also had deteriorated rapidly, but the loss of hearing varied considerably from day to day. He was discharged from the Navy in October 1945, with a 10 per cent disability rating because of deafness. He entered the Mayo Clinic in August 1946, and an audiogram was made (chart 1*b*).

The patient received daily intravenous injections of a histamine phosphate solution from August 5 to 31, without showing any evidence of improvement. On August 31, twenty-six days after treatment was started, the audiogram first showed evidence that the hearing of the right ear had improved (chart 1*c*). The patient continued to receive daily intravenous injections of the histamine solution until September 6, when he was dismissed with apparently normal hearing in the right ear (chart 1*d*). This patient has had no recurrence of difficulty of hearing in six months. He was told on discharge from the Navy that he would become totally deaf. In 1945 histamine therapy was given primarily for the relief of vertigo, whereas in 1946 it was given daily over a more prolonged period to improve the patient's hearing.

This case is instructive inasmuch as it shows that improvement of hearing may not be shown until after more than three weeks of intensive therapy.

CASE 2.—A man aged 34 began to lose ability to hear six months before he first visited the clinic and within four weeks was completely deaf in the right ear and remained so. The loss of hearing began with tinnitus, which remained constant. Three months before he was examined at the clinic the hearing in the left ear began to fail. He had momentary attacks of dizziness at that time, but no vertigo. He received repeated inflations of the eustachian tubes and thyroid therapy without benefit to his hearing (chart 2*a*).

After three weeks of intravenous administration of a histamine phosphate solution the audiogram revealed improvement in the left ear to ability to hear normal conversational tones (chart 2*b*). He was unable to remain longer for treatment at that time but returned in six months for further treatment. At the time of his return an audiogram revealed no change. Further treatment, although carried on for four weeks, produced no further improvement in hearing. (This case was reported in detail, in 1947, by Hallberg and one of us [B. T. H.].[32])

This case also illustrates the fact that improvement can be best obtained early in the course of the disorder.

CASE 3.—A woman aged 22 was epileptic and had attacks of both grand and petit mal. For two weeks, without obvious cause, her hearing decreased until she could not hear ordinary conversation. She had one episode of mild vertigo without nausea or vomiting, which lasted ten to twenty minutes (chart 3*a*).

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32. Hallberg, O. E., and Horton, B. T.: Sudden Nerve Deafness, Proc. Staff Meet., Mayo Clin. **22**:145-149 (April 16) 1947.

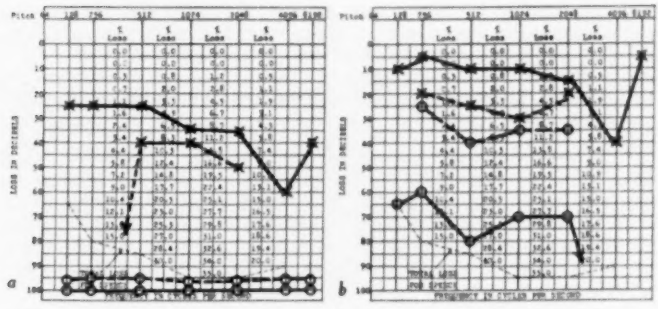

Fig. 2 (case 2).—(*a*) Complete deafness in the right ear and impairment of hearing in the left ear. (*b*) Improvement of hearing in the left ear following three weeks' intravenous administration of solutions of histamine phosphate; the patient could hear normal conversational tones. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line. (From Hallberg and Horton.[22])

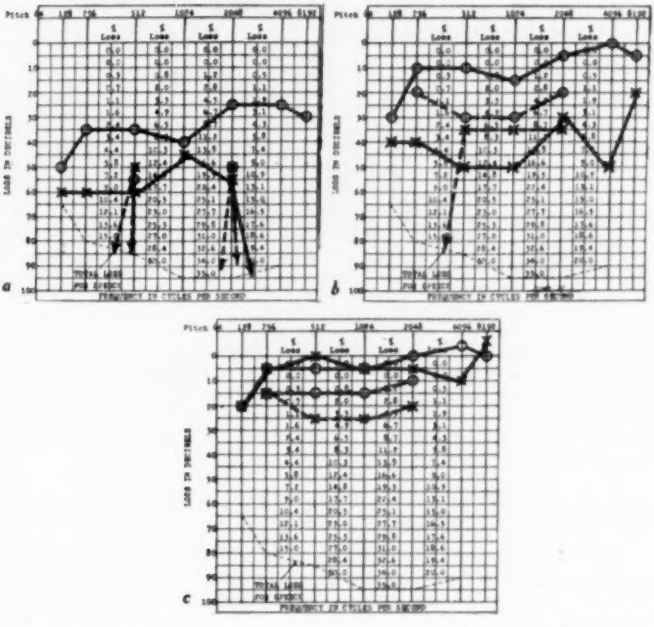

Fig. 3 (case 3).—(*a*) Depiction of patient's inability to hear ordinary conversation. (*b* and *c*) Return of hearing to approximately normal after six daily intravenous injections of a solution of histamine phosphate. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

Hearing returned to normal after a histamine phosphate solution was given intravenously for six days (chart 3 *b* and *c*). Symptoms had not returned nine months later.

This case illustrates the improvement possible in cases in which deafness begins suddenly.

Case 4.—A woman aged 22 had had bilateral tinnitus as long as she could remember. Three years before she was admitted to the clinic her attention was first called to her loss of hearing by the fact that she turned the radio on too loudly for the comfort of others. Deafness had increased steadily. The patient had had vasomotor rhinitis since childhood and had always noted that the tinnitus was worse when the vasomotor rhinitis was severe. A diagnosis of otosclerosis had been made elsewhere (chart 4 *a*).

Because this patient was known to have an allergy, it was decided to give the intravenous use of histamine a clinical trial for approximately two months. After three weeks a slight improvement of her ability to hear low tones was

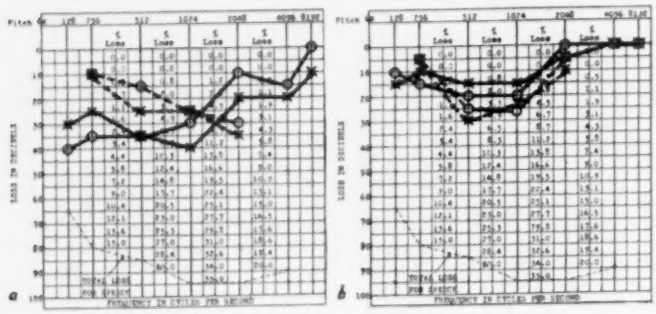

Fig. 4 (case 4).—(*a*) Audiogram of a patient with what had been believed to be otosclerosis. (*b*) Audiogram made after four weeks' intravenous administration of a histamine phosphate solution. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

apparent. A week later an improvement of hearing for all frequencies had taken place, and the tinnitus had stopped entirely (chart 4 *b*). Although treatment was continued for three weeks longer, no further improvement was obtained. This patient was dismissed with instructions to take 0.3 cc. of a 1:10,000 solution of histamine phosphate if symptoms recurred. Tinnitus recurred on several occasions but was relieved by subcutaneous injection of the histamine solution.

This case suggests that sometimes when a diagnosis of clinical otosclerosis is made endolymphatic hydrops may be present.

Case 5.—A man aged 52 noted tinnitus for ten years and deafness in his left ear for a month before he came to the clinic. The impairment progressed steadily. A histamine phosphate solution was given intravenously for fifteen days without benefit (chart 5).

In this case deafness probably was due to atrophy of a nerve as a result of arteriosclerosis.

CASE 6.—A woman aged 45 had a sudden feeling of fulness in the right ear, and in three days hissing tinnitus was present and deafness developed. During the following two weeks hearing improved considerably (chart 6). Hissing tinnitus, the so-called white sound, continued. These symptoms had been present for nine months prior to admission to the clinic. (This was reported in more detail by Hallberg and one of us [B. T. H.][22]).

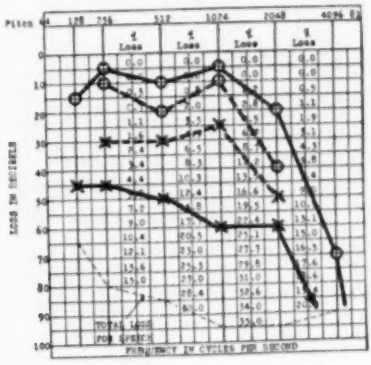

Fig. 5 (case 5).—Audiogram showing failure of hearing to return to normal after fifteen daily intravenous injections of a solution of histamine phosphate. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

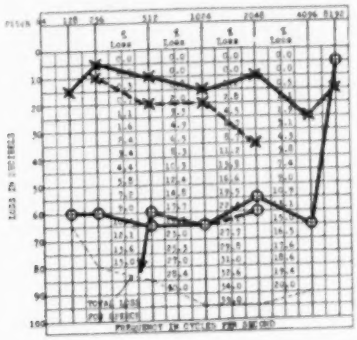

Fig. 6 (case 6).—No improvement in hearing occurred in the right ear after several weeks' treatment with histamine. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

Histamine phosphate was given for thirty-two days without effect. In this case the deafness probably was due to a vascular lesion.

Cases 5 and 6 illustrate the fact that in many cases deafness of sudden onset is probably on a basis other than endolymphatic hydrops.

CASE 7.—Four months before a woman aged 27 came to the clinic, severe pounding tinnitus, sense of fulness in the head and deafness in the right ear developed. She stated that these symptoms had started the night her husband returned home on leave after six months in the armed forces. At the time she was first examined at the clinic she was found to be pregnant, and the symptoms referable to the ear were attributed to this condition. Delivery did not diminish the symptoms, and when the patient returned eight months later, the symptoms were unchanged (chart 7 *a*).

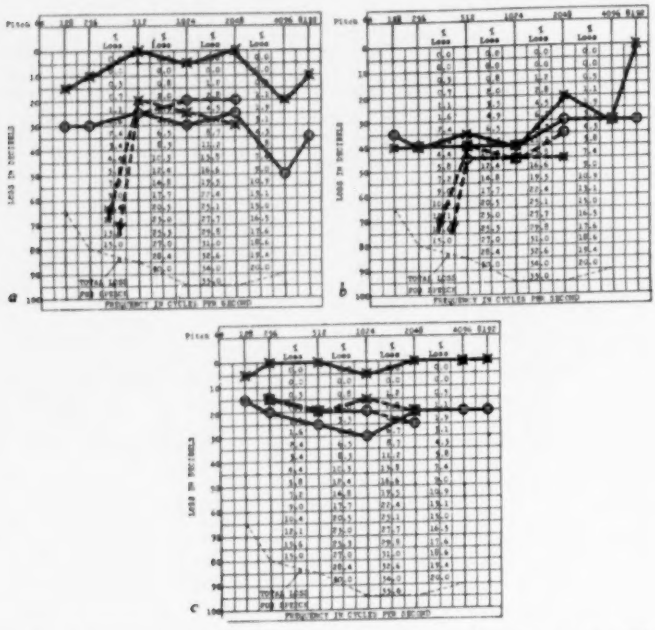

Fig. 7 (case 7).—(*a*) Diminished hearing as manifested at the time of admission of the patient; delivery of a child had not improved hearing. (*b*) Loss of hearing involving both ears. (*c*) Return of hearing in the left ear to approximately normal and marked improvement of hearing in the right ear after intravenous injections of solutions of histamine phosphate and administration of diethylstilbestrol. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

Nicotinic acid was given, and this relieved her symptoms and improved her hearing. After two weeks the patient wrote that the symptoms had recurred. On her return intravenous administration of a histamine phosphate solution was begun. After an initial improvement the symptoms recurred and deafness increased. The tinnitus and the loss of hearing involved both ears (chart 7 *b*). The history and the audiograms were reviewed and it was discovered that the increase of symptoms and deafness occurred in the two weeks before, during and after every menstrual period.

At the suggestion of one of us (L. A. D.), after consultation, the patient was given 0.1 mg. of diethylstilbestrol daily. It was found that administration of this drug alone would not produce or maintain improvement in her symptoms or hearing. With the combined use of histamine (intravenous) and diethylstilbestrol, however, the hearing in the left ear returned to normal and the hearing in the right ear improved (chart 7 *c*).

Case 8.—A boy 11 years old was brought to the clinic because he had strabismus, and deafness was discovered by chance during the routine examination. Hypertrophied tonsils and adenoids were noted (chart 8 *a*).

Tonsillectomy and adenoidectomy were performed, after which radium was applied to the nasopharynx. Subjectively he noted no improvement in hearing following these procedures. A month later, intravenous administration of a histamine phosphate solution was started. Unfortunately, another audiogram was not taken before the histamine was given. The patient's hearing seemed to improve slowly, and after fourteen injections an audiogram was taken (chart 8 *b*).

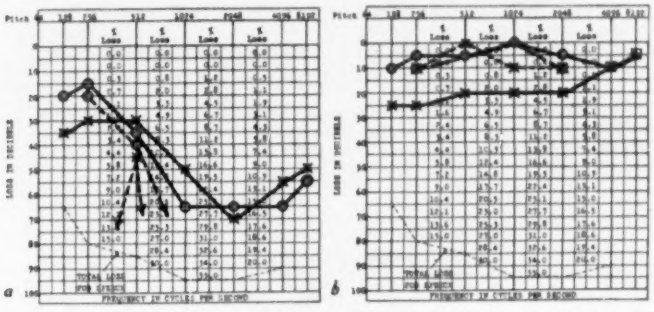

Fig. 8 (case 8).—(*a*) Loss of hearing discovered at routine examination and checked by audiogram. (*b*) Improvement of this patient's hearing after tonsillectomy and adenoidectomy, postoperative application of radium to the nasopharynx and a course of intravenous injections of histamine phosphate solutions. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

Two weeks later after ten more intravenous injections of the histamine solution the hearing was normal in both ears, as shown by audiometry.

The improvement was somewhat unexpected. Although the bone conduction curve was not that anticipated in cases of endolymphatic hydrops, it is felt that the hearing did not improve until administration of histamine was started. Nevertheless, it is possible that the improvement resulted from the postoperative use of radium rather than from the use of histamine. The diagnosis of endolymphatic hydrops cannot be considered as more than tentative in this case.

Case 9.—A man aged 54 had a severe attack of nausea and vomiting four years before he was admitted to the clinic. He had not felt well after that. He thought his hearing was slightly impaired but did not know for how long the impairment had been present. He was found to have moderately severe, diffuse arterial disease with hypertension. The systolic blood pressure was 205 and the

diastolic 125 mm. of mercury. Audiograms taken before treatment showed deafness, which we could not be certain was representative of hydrops (chart 9 *a*).

The hearing in the right ear slowly improved when a histamine phosphate solution was given intravenously daily for three weeks (chart 9 *b*).

According to the audiogram, this patient's hearing made an average gain of 15 decibels for the frequencies 256, 512 and 1024. After the first three weeks,

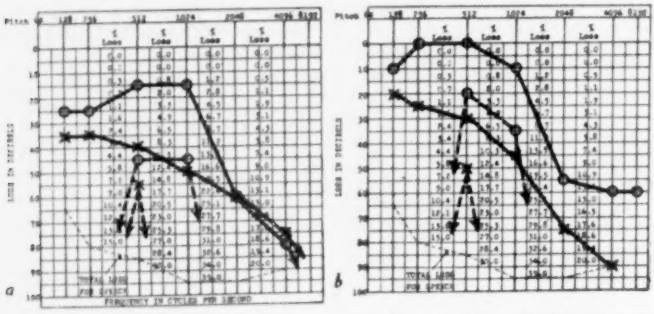

Fig. 9 (case 9).—(*a*) Loss of hearing present on admission. (*b*) Improvement of hearing in the right ear after about three weeks' intravenous administration of a solution of histamine phosphate. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

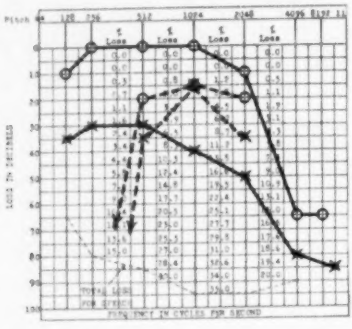

Fig. 10 (case 10).—Audiogram made three years after treatment; no improvement of hearing. Treatment consisted of intravenous administration of histamine phosphate for six days. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

although he received thirty additional injections, no further improvement was shown.

In this patient the diagnosis of the type of deafness present was extremely difficult to establish but the deafness was probably due to the diffuse arterial disease.

Case 10.—A man 49 years old stated that he had suffered from severe unilateral headaches most of his life. Some dizziness, but no vertigo, had accom-

panied the severe migraine. He had had tinnitus in the right ear for fifteen years and deafness in the left ear for an indefinite period. He had severe astigmatism, never corrected by glasses, for he would not wear them. He had sclerosis, grade 1, of the retinal arterioles. His blood pressure was 170 systolic and 98 diastolic.

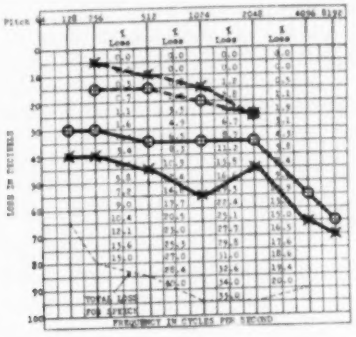

Fig. 11 (case 11).—Audiogram revealing that there was no effect on the loss of hearing from intravenous injections of solutions of histamine phosphate. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

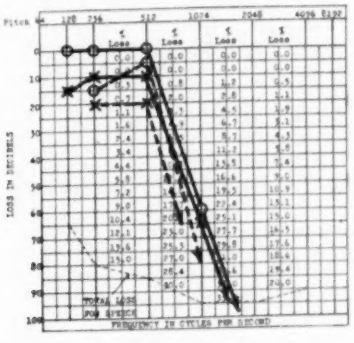

Fig. 12 (case 12).—Audiogram demonstrating that there was no return of hearing after ingestion of large doses of vitamins and intravenous injection of a thiamine hydrochloride solution. Key: Right ear, *o*; left ear, *x*; air conduction, solid line; bone conduction, broken line.

This patient was given a histamine phosphate solution intravenously daily for one week. No change in symptoms or in audiogram occurred (chart 10).

Cases 9 and 10 illustrate the fact that all deafness occurring in vagotonic persons cannot be classed as that due to endolymphatic hydrops.

Case 11.—A physician aged 35 came to the clinic in 1943 because of severe headaches of the histaminic cephalgia type, which had ceased spontaneously six months previously. He wanted to prevent future attacks. Before the headache developed, he had vasomotor rhinitis and myalgia on exposure of the skin surface to change in temperature for many years. An audiogram taken in 1945 (chart 11) showed essentially the same picture as one taken in 1943. Twenty-three intravenous injections of a histamine phosphate solution had no effect on hearing.

Case 12.—A woman aged 50 was in a Japanese prison camp for two and a half years and lost 40 pounds (18.1 Kg.). She had weighed 130 pounds (59 Kg.). During this time she had considerable loss of vision, which later was partially regained, and loss of hearing, which did not improve.

She was given large doses of vitamins by mouth, and, in addition, a thiamine hydrochloride solution was given intravenously, but symptoms did not decrease (chart 12). It was thought that in this case atrophy of the organ of Corti had occurred as the result of avitaminosis.

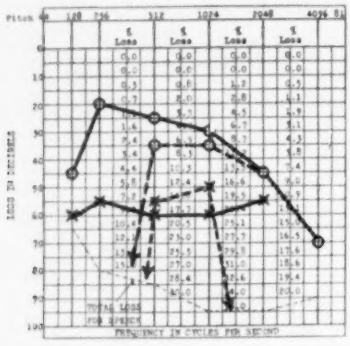

Fig. 13 (case 13).—Audiogram typical of nerve deafness. It is a senile type of deafness not amenable to treatment. Key: Right ear, *o;* left ear, *x;* air conduction, solid line; bone conduction, broken line.

Case 13.—A man aged 76 had noticed that deafness was gradually increasing for ten years before he was admitted to the clinic. It was a senile type of deafness.

The audiogram in this case (chart 13) was the typical audiogram of nerve deafness, and the condition probably was caused by degeneration of the cells of the organ of Corti. This type of deafness is not amenable to treatment.

COMMENT

The group of 32 cases studied, we believe, represents an attempt to separate from the larger group of cases of nerve (inner ear or perceptive) deafness a subgroup in which the hearing may be restored by treatment. We believe that the loss of hearing in this group is due to endolymphatic hydrops, which produces the classic symptoms of vertigo and nausea.

We submit that certain points in the history and certain observations can help distinguish the group with endolymphatic hydrops from the general group with inner ear deafness. The points are a history suggestive of an allergic (pathergic) background, frequently an audiogram giving evidence of typical deafness for low tones, especially noticeable in the bone conduction curve, and the presence of binaural diplacusis in those in whom the hearing of the two ears differ considerably.

We have further indicated a method of treatment which has produced a certain measure of restoration of hearing in many of these patients.

ABSTRACT OF DISCUSSION

DR. LAWRENCE BOIES, Minneapolis: The essayist suggests as criteria for making a tentative diagnosis of endolymphatic hydrops in the absence of vertigo the following four considerations:

1. Hearing loss of low tones, with high tones more or less preserved. I should expect this to apply only to cases in which the impairment was in an early stage.

2. A history of fluctuating loss; in women, a temporary loss associated with menses.

3. Certain allergic phenomena occurring in association with the impairment of hearing (vasomotor rhinitis, urticaria, dermatitis factitia, severe unilateral headaches).

4. A sudden decrease in ability to hear, occurring in two or three days, with persistence of the impairment.

Dr. Williams pieces together the observations of a number of observers concerning the phenomena associated with physical allergy to provide a plausible basis for his hypothesis that "endolymphatic hydrops is a form of physical allergy probably involving the capillary loops of the stria vascularis" and that "it is improbable that the picture of extracellular edema can be produced in the body by any agency other than allergy."

His inquiry extends into the field of endocrinology. A number of observers have noted the effect of the endocrine glands on the release of histamine. The essayist suggests that in women presenting evidence that certain endocrine disturbances are associated with their menses, and especially in those who complain of tinnitus and dizziness and, in some instances, of impairment of hearing, an abnormal permeability of capillary walls has resulted in endolymphatic hydrops, and that this is the foundation for the aural symptoms. He has learned that administering diethylstilbestrol in combination with histamine may improve the hearing of these women.

The essayist has recognized the possibility that psychogenic deafness may influence his results and seemingly has excluded that factor from the 32 cases in which a diagnosis of endolymphatic hydrops without vertigo was made.

I have already stated that it is my belief that this paper will stimulate a lot of thought. It will also stimulate a lot of therapy. Some of it will be indiscriminate, but that is no discredit to the essayist. It has been my observation that the advent of antihistaminic substances into the field of otolaryngology has supplied a lot of ready ammunition for those who seem to be strong on therapy and, at times, short on diagnosis.

Dr. Williams has indicated that the diagnosis of endolymphatic hydrops without vertigo as a cause of losses in hearing will be an uncommon one. This emphasizes the point that otologists must be aware of all of the other uncommon causes of hearing losses. The following experience illustrates this point:

A 55 year old man was referred to me recently with a history of gradual impairment of hearing in his left ear over a four year period. A low-pitched tinnitus had developed after he had become aware of the deterioration of his hearing. He had never experienced any vertigo. He gave a history of having received "a course of histamine and some shots of nicotinic acid." The usual inspection of his ear drums revealed nothing significant.

Tuning fork tests supported a diagnosis of a perceptive type of loss. If this man had been experiencing vertiginous attacks, I should have promptly made a tentative diagnosis of Ménière's disease involving the left labyrinth.

The patient was a willing subject for any therapy which one might suggest. I considered the idea of giving him intensive and prolonged histamine therapy to see what would happen. My experience had led me to believe that this is necessary for substantial results. However, I decided to first have him more thoroughly studied. The consulting neurologist found the corneal reflex decreased on the left side, the biceps and triceps reflexes more active on that side and the signs positive on the right side. Roentgenograms showed an increase in the size of his left internal auditory meatus and decalcification or erosion of the petrous tip on that side. The patient now has a date with a neurosurgeon.

It was in 1861 that Prosper Ménière first described the symptoms of the clinical entity which has borne his name. Seventy-seven years elapsed before Hallpike and Cairns in 1938 first described the pathologic changes observed in a labyrinth affected with endolymphatic hydrops. Since then it has been frequently remarked that though physicians know what occurs in the labyrinth in this disorder, the actual etiologic factor is still obscure. Today otolaryngology may have turned another corner.

PRESIDENT GROVE: This paper is now open for general discussion. Dr. Macfarlan!

DR. DOUGLAS MACFARLAN, Philadelphia: Dr. Williams spoke about the factor of psychogenic deafness in this study. It may be a rather mean question to ask any one, but—how does he test for psychogenic deafness?

DR. CHARLES E. KINNEY, Cleveland: This brings out once more the necessity for the practicing clinical otologist to have handy a rather rapid method for testing for impairment of hearing of discrete frequencies and of speech. That testing cannot be postponed two or three days, because many of these phenomena are then gone.

DR. ARTHUR M. ALDEN, St. Louis: Dr. Williams' presentation is a splendid example of good otologic thinking. He has been able to separate out from the diagnostic heap which otologists hitherto called perception deafness of undetermined origin, and about which they have been able to do little or nothing, at least a group of cases about which he has been able to do something.

What he has been able to accomplish in this relatively small group of cases to me is less important than the establishment of this line of thinking about them.

PRESIDENT GROVE: Is there any further discussion? If not Dr. Williams, will you close the discussion?

DR. HENRY L. WILLIAMS, Rochester, Minn.: In answer to Dr. Macfarlan's question about the test for psychogenic deafness: If we have any suspicion of

psychogenic deafness, we usually send the patients to a psychiatrist. We have not used partial anesthesia, as Semenov has advised, but have depended on other criteria to separate these patients. In none of the patients was there a sudden return of hearing. It always returned slowly. In none of the patients was there any evidence of instability as far as their personality was concerned. It may well be that some cases of psychogenic deafness have been included, but I think not.

Regarding Dr. Kinney's remarks about the necessity for rapid testing, I believe one necessity in helping these patients is to see them over a long period, so that one can follow them rather closely and have them come for testing whenever their hearing drops. Of course, the necessity of keeping them around for such a long time for treatment is something of a nuisance.

Fortunately, with Dr. Horton we have a setup such that the otologist can turn these patients over to him for treatment. I am certainly grateful for Dr. Horton's collaboration.

Dr. Berry has asked us if we have used this treatment in deafness resulting from pregnancy. We have not used it. Of course deafness from pregnancy is often associated with the toxemias of pregnancy and apparently is due to the same capillary disorder which has been demonstrated by Müller to be present in Ménière's disease or in endolymphatic hydrops.

ALLERGY OF THE EAR

With Classification and a New Treatment of Labyrinthine Hydrops by Dehydration

ARTHUR DINTENFASS, M.D.
ATLANTIC CITY, N. J.

ALLERGIC conditions of the ear and pathologic variations due to altered sensitivity occur more frequently than generally recognized. A review of the various allergic manifestations with some attempt at clarification and classification should be of value to the otologist and allergist. It is now a well established fact that allergy predisposes to and aggravates many diseases of the upper respiratory tract. Recent surveys, including those of Shambaugh,[1] King,[2] and Hamlin [3] relating to the frequency of allergy as an etiologic factor or as a complication in diseases of the ear, nose and throat, show 70 to 90 per cent of all office patients to be allergic. Still more recently there has been interest in the relation of allergy to the ear. Jones [4] stated that allergic reactions involving the mucous membrane of the eustachian tube and tympanic cavity have not received sufficient consideration.

The primary factor in the clinical aspect of the allergic reaction and the fundamental concept with which the otologist is concerned is that the various parts of the ear—external and middle, together with the eustachian tube and the region about its orifice, and the inner ear—be considered a shock organ. It is at these sites that the allergic reaction takes place after the exposure of the tissue antibody to the antigen.

A preliminary presentation of this study was made at a meeting of the Philadelphia Laryngological Society, May 1947.

From the Otolaryngology Department, University of Pennsylvania Graduate School of Medicine, and the departments of Allergy and Otolaryngology, Atlantic City Hospital.

Thesis approved by the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science [M.Sc. (Med.)] for graduate work in otolaryngology.

1. Shambaugh, G. E., Jr.: Nasal Allergy for the Practising Rhinologist, Ann. Otol., Rhin. & Laryng. **54**:43-60 (March) 1945.

2. King, E.: Why Otolaryngologists Should Be Allergy Minded, Ohio State M. J. **40**:138-140 (Feb.) 1944.

3. Hamlin, F. E.: An Investigation of Allergy in Routine Nose and Throat Practice, Virginia M. Monthly **73**:45-50 (Feb.) 1946.

4. Jones, M. F.: Manifestations of Allergy in the Ear, Ann. Otol., Rhin. & Laryng. **47**:910-916 (Dec.) 1938.

The following classification is presented in an endeavor to simplify and relate the various allergic manifestations of the ear:

CLASSIFICATION OF ALLERGY OF THE EAR

Location	Signs and Symptoms
External ear and auditory canal	1. Contact dermatitis 2. Eczema 3. Urticaria 4. Purpura
Middle ear and eustachian tube Primary Secondary to nasal allergy	1. Conduction deafness 2. Clinical otitis media 3. Sensation of fulness 4. Itching in the ear, back of the nose or between the nose and the ear 5. Deep pain—dull or burning 6. Sensation of tightness or drawing
Internal ear and auditory nerve	1. Perceptive deafness 2. Nystagmus 3. Ménière's symptom complex 4. Vertigo 5. Tinnitus

The classification is made on an anatomic basis. The main divisions are on the external ear and the auditory canal; the middle ear and the eustachian tube, and the internal ear and the auditory nerve. For practical purposes, the subdivisions are concerned with clinical findings.

EXTERNAL EAR

The skin of the external ear is subject to the same allergic conditions as the skin elsewhere on the body, in addition to those conditions which occur because of the location and structure of the ear. In my experience, I have found that otitis externa due to allergic factors exists more frequently than is usually suspected or diagnosed. This condition in the greatest percentage of cases has been found to be due to contact sensitivity; however, ingestants, such as food and drugs, may produce eczema, urticaria and purpura.

Hansel [5] noted the predominance of allergic otitis externa and stated that most of nonallergic external lesions are the result of fungous infections or discharge from the middle ear.

Contact dermatitis of the ear has been activated by cosmetics, clothing, bedclothes, drugs, bacteria, plants and plant pollen. Because of

5. Hansel, F. K.: Allergy in Otolaryngology and Ophthalmology: Review of Recent Current Literature, Ann. Allergy **2**:165-172 (March-April) 1944.

the accessibility of the ear to the environment, as well as to the fingers, with repeated manipulations at all ages, the frequency of aural dermatitis is readily understandable.

Cosmetics are a common source of epidermal sensitivity, including face powder and creams, rouge, lipstick, finger-nail polish, hair dyes, wave-set lotions and perfumes. The causative factors of epidermal sensitivity in these preparations are orris root, vegetable gum bases, dyes and synthetic compounds.

Any of the usual and many of the unusual forms of clothing have produced allergic skin conditions of the ear. Woolens, silks, rayon and nylon are among the offenders. Fur neckpieces and collars are not infrequent sources of reactions about the ear. Metal and composition spectacle bows occasionally produce irritations in this region. Otitis externa has been known to disappear when pillows containing feathers, horsehair or kapok have been removed from the bed.

Vaughan [6] mentioned sensitivity to telephone and other hard rubber ear phones. No definite sensitivity to acrylic resin base synthetics, such as used in ear pieces for hearing aids, has as yet been reported, although 2 patients with such sensitivity have recently been seen at the Philadelphia Naval Hospital.[7] One should keep in mind these methyl methacrylate prostheses as potential sensitizers.

Many of the drugs used locally in treating otitis externa and media cause allergic skin reactions. The sulfonamides, mercurials and other heavy metals, as well as phenols and cresylic acid, may be offenders. Consequently, it is well to be aware that active medication may be producing symptoms when there is an exacerbation under treatment or when the patient does not respond to therapy.

REPORT OF A CASE OF ALLERGIC REACTION TO MEDICATION

CASE 1.—A white woman aged 64 was seen on Dec. 8, 1948 with complaints of swelling, pain and discharge from both ears. The past history included allergic conjunctivitis, pruritus ani and rhinitis, as well as a family history consistent with allergy. The onset of the present condition occurred on November 30, with symptoms of itching of the ears, followed by discharge, a slight decrease in hearing and mild pain. A physician was consulted and various types of medications were used without success. On December 6 a preparation containing sulfanilamide was applied to the external auditory canal with the aid of a gauze wick. When the patient was seen on December 8, she complained of excruciating pain referred to the ears and adjacent areas.

Examination showed an acute inflammatory reaction involving the entire external ears, spreading to the face, head and neck. Both external auditory canals were so edematous that an applicator could not be admitted.

6. Vaughan, W. T.: Practise of Allergy, revised by J. H. Black, ed. 2, St. Louis, C. V. Mosby Company, 1948.

7. Sataloff, J.: Personal communication to the author, April 1949.

The patient responded rapidly to thorough cleansing, local applications of warm boric acid solution and the avoidance of medication containing sulfonamide derivatives.

Comment.—This patient apparently had been sensitized to the sulfonamide by previous ingestion. The exacerbation is considered an allergic reaction because the clinical relation to the application of the allergen was definite, the response to its removal prompt and because of the personal and family history of allergy.

Occasionally, a bacterial allergy exists to the streptococci, staphylococci or fungi which inhabit the external auditory canal in association with cerumen and epithelial debris.[8]

Dermatitis venenata of the ear occurs after contact with certain plants, such as poison ivy, poison oak and primrose. Other essential oils found with grass and weed pollens give rise to an obscure type of seasonal dermatitis, which is usually associated with lesions on other parts of the body.

Eczema and eczematoid lesions predominate in young children in conjunction with more or less generalized lesions of the head and flexor surfaces and are usually caused by common ingredients of the diet, such as eggs, wheat, milk, oranges and chocolate, subsiding when the necessary dietary adjustments are made. However, as Livingood and Pillsbury[9] have emphasized, when local therapy is used, the sulfonamides may aggravate and prolong eczematous conditions.

MIDDLE EAR

As shown in the foregoing classification, the middle ear is concerned with a variety of clinical findings that have been attributed to allergy. There is quite a diversity of opinion among authorities as to the frequency. Proetz[10] stated that allergic reactions of the middle ear are more prevalent than is generally known and are responsible for many a needless and futile myringotomy. To facilitate the understanding of this category, I have made a division into primary allergic states of the middle ear and those secondary to nasal allergy, by reason of anatomic association through the eustachian tube.

The typical case[10] of primary middle ear allergy is one in which there is history of intense pain, roaring and partial deafness of one or both ears, occurring suddenly after exposure to or ingestion of some foreign protein. The drum membrane is red and swollen, but does

8. Williams, H. L.: Montgomery, H., and Powell, W. N.: Dermatitis of the Ear, J. A. M. A. **113**:641-646 (Aug. 11) 1939.

9. Livingood, C. S., and Pillsbury, D. M.: Sulfathiazole in Eczematous Pyoderma, J. A. M. A. **121**:406-408 (Feb. 6) 1943.

10. Proetz, A. W.: Allergy in the Middle and the Internal Ear, Ann. Otol., Rhin. & Laryng. **40**:67-76 (March) 1931.

not have the typical convexity of infectious otitis media. Often the drum membrane has a whitish cast, due to edematous epithelium, and is similar in appearance to the membrane of the allergic nose. Incision gives a sensation of cutting into a spongy structure instead of a taut membrane. It is to be emphasized that most cases occur in children under 5 years, and the severest cases occur among recently weaned infants who are sensitive to some new food in their diet.

There are an impressive number of reports in the literature showing definite relation between allergy and diseases of the middle ear. Lewis [11] was among the first to point out that it was possible for acute otitis media to be of allergic origin. He presented 6 cases in which there was spontaneous serous discharge, preceded by severe earache, with pronounced impairment of conduction hearing. The discharge ceased in thirty-six to seventy-two hours, with prompt closure of the perforation and restoration of hearing. It is conceivable that many cases of acute otitis media originate in this manner and become infectious as a result of secondary bacterial invasion. Felderman [12] reported several cases of catarrhal otitis media, complicated by mastoiditis, in which decided improvement occurred when the causative foods, egg and milk, were eliminated from the diet. Jones [4] discussed a similar condition in a 7 year old child, in whom symptoms were produced by the ingestion of nuts. Noun [13] described 2 cases of chronic otorrhea in which apparent cure resulted from careful selection of food. Koch [14] studied 222 patients with chronic otitis media and found that approximately 20 per cent presented an eosinophilic discharge. This condition was associated with an edematous appearance of the mucosa of the ear and allergic rhinitis. Allergic studies and therapy were effective in healing more than one half of these patients. A review of the past histories of personally observed patients with infections of and operation on the mastoid process indicated that fully 50 per cent have a background of allergy.

Otitis media may occur secondary to nasal allergy in conjunction with allergic rhinitis or hay fever of the seasonal type. It also may be secondary to isolated edema of the eustachian tube by mechanical occlusion. Such involvement may cause a feeling of stuffiness in the head, impaired hearing and pain or may produce a clinical picture identical with that of the usual secretory or catarrhal otitis media.

11. Lewis, E. R.: Otitis Media and Allergy, Ann. Otol., Rhin. & Laryng. **38**:685-690 (Sept.) 1929.

12. Felderman, L.: Allergy of the Upper Air Passages: Inherited and Acquired Factors, Ann. Otol., Rhin. & Laryng. **45**:80-100 (March) 1936.

13. Noun, L. F.: Chronic Otorrhea Due to Food Sensitivity, J. Allergy **14**:82-86 (Nov.) 1942.

14. Koch, H.: Allergic Investigations of Chronic Otitis, Acta oto-laryng. 1947, supp. 62, pp. 1-201; abstracted Arch. Otolaryng. **47**:533-534 (April) 1948.

REPORT OF A CASE OF ACUTE ALLERGIC OTITIS MEDIA OBSERVED AT THE ONSET OF TWO CONSECUTIVE SEASONS OF RAGWEED HAY FEVER

CASE 2.—A white woman aged 32 was seen at 1:30 a. m. on Aug. 16, 1947 with complaints of severe pain and discharge from the left ear. This patient presented a personal history of seasonal and perennial allergic rhinitis, together with a family history of asthma, allergic rhinitis, migraine and various types of skin allergy.

The patient had been perfectly well until approximately two hours before the onset of aural pain, when she had had a severe attack of nasal congestion, sneezing and rhinorrhea. Within one-half hour of the onset of symptoms, pain in the left ear became apparent and increased rapidly in severity. At 1 a. m. there was a sudden outpouring of clear fluid from the left ear, associated with an appreciable decrease in pain.

Examination disclosed a perforation about ⅛ inch (0.32 cm.) in diameter in the anterior superior quadrant of the left tympanic membrane. The remainder of the drum membrane was dull with a whitish cast. The discharge was clear, somewhat tenacious and without odor. The nasal membranes and turbinate bodies were exceedingly engorged, with the typical appearance of allergic rhinitis. A smear was taken of the discharge and found to have an approximately 50 per cent eosinophilic content.

On Aug. 17, 1948, one year later, the patient experienced a similar attack, with pain in the left ear following severe nasal symptoms.

Examination revealed diffuse bulging of the left tympanic membrane, with a dull edematous appearance, and severe allergic rhinitis. Conservative treatment of the nose, orifices of the eustachian tube and middle ear, without rupture or incision of the drum membrane, resulted in subsidence of the symptoms. The patient was afebrile during the course of both attacks.

Comment.—This case is a typical case of allergic otitis media, with two attacks occurring at the onset of the season of ragweed hay fever in a patient of definite allergic predisposition and clinical manifestations, whose family history is replete with allergic diseases.

Jones [4] and Dean [15] described the characteristic appearance of the nasopharynx in nasal allergy associated with aural symptoms. The orifice of the eustachian tube is edematous, and the mucous membrane of the posterior septum is pitted and is bluish gray. A fold of mucous membrane, similar in appearance to a false membrane, overlies the septal mucosa. The condition is frequently bilateral and occasionally is of such intensity as to occlude the air passages partially. A profuse watery discharge from the tympanum after a myringotomy probably results from blockage of the eustachian tube by this process.

INNER EAR

The symptoms attributed to allergy of the inner ear and auditory nerve vary greatly in intensity. They range from clicking sounds, isolated tinnitus, slight vertigo and nystagmus to a clinical picture that

15. Dean, L. W., Jr.: Ménière's Syndrome and Allergy, Internat. Clin. **3**:231-234 (Sept.) 1941.

includes the severest attacks of rotary vertigo, intense nystagmus, disappearance of the caloric reaction, pronounced tinnitus and definite impairment of hearing.

The inner ear mainly is affected by Ménière's symptom complex. In the classic case there is sudden onset of the triad of deafness, tinnitus and vertigo, associated with disturbance of equilibrium, spontaneous nystagmus and, frequently, nausea and vomiting. These attacks are paroxysmal and last from a few moments to several days or longer. Repeated attacks result in increased loss of function.

It is the opinion of many authorities [16] that allergic reactions in the labyrinth are responsible in a significant percentage of cases for the symptoms which constitute this syndrome. These allergic reactions are responsible to a less degree for isolated vertigo, tinnitus and deafness.[17] It is characteristic that when these individual findings are due to allergy they vary from day to day or from week to week without objective changes in the tympanic membrane. Some investigators find that these symptoms respond readily to injection with epinephrine.[18]

A number of reports indicate the close relation of Ménière's syndrome to allergy. In 1893 Quincke [19] expressed the belief that the pathologic process was similar to that found in angioneurotic edema. Descriptions of the coexistence of Ménière's symptom complex with urticaria or serum disease and with other allergic manifestations, such as hay fever and asthma, are too numerous to be coincidental.[20] A definite relation was found in a patient (case 3) with Ménière's syndrome, whose initial symptom was urticaria following ingestion of pork and chocolate. Sensitivity to these items was confirmed by skin tests. Duke [21] and Rowe [22] observed this syndrome in patients with severe allergy for whom

16. (*a*) Feinberg, S. M., and Durham, O. C.: Allergy in Practice, Chicago, The Year Book Publishers, Inc., 1944. (*b*) Williams, H. L.: Intrinsic Allergy as It Affects Ears, Nose and Throat, Ann. Otol., Rhin. & Laryng. **53**:397-443 (Sept.) 1944; The Present Status of the Diagnosis and Treatment of Endolymphatic Hydrops (Ménière's Disease), ibid. **56**:614-646 (Sept.) 1947. (*c*) Hansel.[5] (*d*) Lewis.[11] (*e*) Dean.[15]

17. (*a*) Criep, L. H.: Allergic Vertigo, Pennsylvania M. J. **43**:258-262 (Dec.) 1939. (*b*) Fuchs, A. M., and Almour, R.: Allergic Deafness, New York State J. Med. **47**:1397-1398 (June 15) 1947. (*c*) Lewis.[11]

18. (*a*) Kuhn, H. A.: Allergy in Hearing Defects, J. Indiana M. A. **36**:143-147 (March) 1943. (*b*) Dean.[15]

19. Quincke, H.: cited by Feinberg.[16a]

20. Vogel, K.: The Relation of Ménière's Disease to the Allergic Disposition and to Other Irritations of the Vegetative Nervous System, Arch. Otolaryng. **15**:322 (Feb.) 1932. Hansel.[5] Vaughan.[6] Dean.[15]

21. Duke, W. W.: Ménière's Syndrome Caused by Allergy, J. A. M. A. **81**:2179-2181 (Dec. 29) 1923.

22. Rowe, A. H.: Food Allergy: Its Manifestations, Diagnosis and Treatment, with a General Discussion of Bronchial Asthma, Philadelphia, Lea & Febiger, 1931.

relief was obtained by the use of epinephrine and avoidance of the allergens, the symptoms being reproduced during well periods by ingestion of certain foods. Orris root, as an inhalant in powder form, has produced Ménière's syndrome when sensitive persons attended barber shops, theaters, dances and crowded places.[23] Lermoyez' syndrome, which differs from Ménière's syndrome in that both tinnitus and deafness precede the vertigo and diminish or completely subside immediately after the vertigo becomes established, is stated by Eagle [24] to have an allergic basis, with urticaria as the essential etiologic factor.

My experience in a large Army hospital, in which I specialized in neurologic disorders, lends clinical support to the concept of allergy of the inner ear. A heterogeneous group of more than 200 patients, with varied symptoms of headache, vertigo, tinnitus and deafness, were studied. All these patients were given skin tests with histamine. Early in the series Atkinson's method [25] was used; later, a smaller dosage was found more desirable. Histamine was administered on a definite schedule for all patients with these complaints. Little correlation was found between the skin sensitivity and the clinical effectiveness of histamine therapy. The best results were obtained in those cases in which a diagnosis of allergy was made and in which allergic measures were used in conjunction with histamine. In this group 2 cases are of sufficient interest to merit special attention. The first is that of a milk-sensitive person who had typical attacks of Ménière's symptom complex when milk or milk products were taken, and the second, that of a soldier in whom vertigo and asthma developed after the ingestion of fresh fruits or wheat.

Criep [17a] noted that a large percentage of patients suffering with allergic headaches or migraine complained of recurrent paroxysmal vertigo, which was traced, in many instances, to sensitivity to various allergens.

The pathogenesis of Ménière's syndrome as an allergic entity has been concerned with definite pathologic evidence and many theories. Hallpike and Cairns,[26] in 1938, were the first to observe distention of

23. Yandell, H.: What Orris Root May Do in the Labyrinth Storm, Southwestern Med. **17**:257-258 (Aug.) 1933. Malone, J. T.: Allergy to Orris Root (Rhizome) Powder as an Etiological Factor in Ménière's Disease, M. Bull. Vet. Admin. **9**:406-408 (April) 1933.

24. Eagle, W. W.: Lermoyez's Syndrome—An Allergic Disease, Ann. Otol., Rhin. & Laryng. **57**:453-464 (June) 1948.

25. Atkinson, M.: Ménière's Syndrome: The Basic Fault? Arch. Otolaryng. **44**:385-391 (Oct.) 1946.

26. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625-655 (Oct.) 1938.

the endolymph system in the temporal bones of 2 patients with Ménière's syndrome who died shortly after operations severing the eighth nerve. Lindsay [27] recently compiled a total of 18 cases of labyrinthine dropsy and added 2 cases of his own. He found that deafness was a constant symptom, and in two thirds of the described cases a diagnosis of Ménière's syndrome was made during life. Cawthorne,[28] after reviewing the literature and studying more than 400 cases, concluded that a firm pathologic basis for the disease has been established and that the essential mechanism is a destructive distention of the endolymphatic system.

The increased quantity of endolymph, with a concomitant increase in endolymph pressure, may result from vasomotor disturbances affecting the capillaries in the stria vascularis or the ductus endolymphaticus. The sequence most likely responsible is vasodilatation with increased permeability of the capillary walls, producing secondary edema of the labyrinth. This is the fundamental mechanism of the allergic reaction. An increased sensitivity is responsible for a certain percentage of cases of Ménière's syndrome. Atkinson [25] employed the knowledge that histamine and histamine-like substances mediate vasodilatation and regarded patients with this syndrome as allergic persons sensitive to histamine. Further, vasoconstriction may produce vasodilatation with increased capillary permeability by primarily causing anoxemia of the vessel walls.

Williams [16b] stated the hypothesis that endolymphatic hydrops is due to physical or intrinsic allergy, which may be a perversion of a normal physiologic reaction—the alarm reaction of Selye. This reaction is mediated by the sympathicoadrenal system via the hormones of the adrenal medulla, causing alteration of the permeability of cell membranes and of water and electrolyte metabolism.

Furstenberg [29] demonstrated that low sodium diet and administration of ammonium chloride produced good results in patients with Ménière's syndrome and claimed support for the theory that angioedema of the labyrinth was responsible. Mygind [30] placed the main emphasis on water balance. He maintained that the syndrome is due to an edema of the labyrinth, and in his cases he severely limits the fluid intake, in addition to administering ammonium chloride.

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27. Lindsay, J. R.: Labyrinthine Dropsy, Laryngoscope **56**:325-341 (July) 1946.

28. Cawthorne, T.: Ménière's Disease, Ann. Otol., Rhin. & Laryng. **56**:18-38 (March) 1947.

29. Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ménière's Symptom-Complex: Medical Treatment, Ann. Otol., Rhin. & Laryng. **43**:1035-1046 (Dec.) 1934.

30. Mygind, S. H., and Dederding, D.: The Pathogenesis of Ménière's Disease and of Kindred Conditions in the Ear and the Rest of the Body, Ann. Otol., Rhin. & Laryng. **47**:938-946 (Dec.) 1938.

I have used meralluride sodium solution (mercuhydrin sodium solution®) [31] to produce more efficient dehydration,[32] as an adjunct to the regimen of Furstenberg and Mygind, and have obtained excellent results.[33] Meralluride is one of the newer and less toxic mercurial diuretics, which contains a sodium mercurial salt combined with theophylline. This preparation is being used extensively as a diuretic in the edema in cardiorenal disease, cardiac decompensation and nephrosis and in the ascites in liver disease. The patients in this series were placed on a low salt diet with limitation of fluids. Ammonium salts were used in the first few cases but not in the later ones. Meralluride sodium solution was injected intramuscularly in doses of 2 cc. every second or third day. Charts were kept of the patient's weight, blood pressure, pulse rate and subjective clinical evaluation of symptoms. Audiometric examinations, when possible, were performed at each visit. The following report is typical of the cases in the series.

REPORT OF A CASE OF MÉNIÈRE'S SYNDROME OF ALLERGIC ORIGIN AND IMPROVEMENT FOLLOWING DEHYDRATION THERAPY WITH MERALLURIDE SODIUM SOLUTION

CASE 3.—A white man aged 38 was first seen on September 1948 with complaints of severe dizziness, associated with falling, tinnitus, difficulty in hearing and nausea. The history of these attacks dated back to May 1945, during the time that the patient was in military service, with a recurrence every two or three months. They usually were of sufficient severity to incapacitate the patient completely for a week or ten days, during which time he had been confined to bed in a prone position. He was studied in military service prior to his discharge in 1945 and since then by several physicians without discovery of an etiologic factor. The frequency and severity of these attacks were uninfluenced by any form of treatment, including injections of various types of vitamin preparations and restriction of salt and fluids.

Examination of the patient on September 13 disclosed nothing essentially abnormal with the exception of mild retraction and thickening of both tympanic membranes. Tuning fork tests showed bilateral mixed deafness. Audiometric studies (chart 1) performed during a quiescent phase of the symptoms showed moderate bilateral decrease for the low tone and conversational ranges and slight decrease for the higher tones. The left ear was affected more than the right. The caloric test showed hypoactivity of the left labyrinth.

Skin tests for allergy were performed by the scratch method when the information was obtained that the patient had hives for several days prior to each attack. The reactions were positive for the following items:

Inhalants: Camel hair, cattle hair, dog hair, human hair, wool, mixed feathers, seal fur, cigaret smoke, coconut fiber and orris root.

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31. Mercuhydrin sodium solution® was supplied by the Lakeside Laboratories, Inc., Milwaukee. Gold, H.: Pharmacologic Basis of Cardiac Therapy, J. A. M. A. **132**:547-554 (Nov. 9) 1946.

32. All patients with labyrinthitis, including those with Ménière's syndrome, are being studied in this series.[33]

33. Dintenfass, A.: Dehydration Therapy of Labyrinthine Dysfunction with Mercuhydrin, to be published.

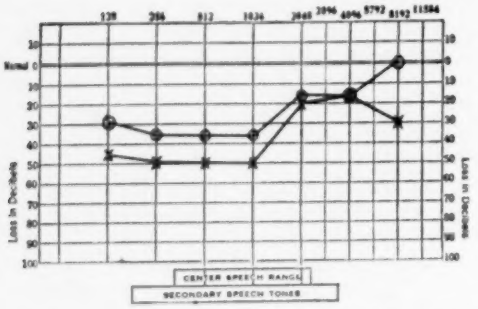

Chart 1.—Audiogram made during quiescent phase of symptoms in case 3. The decibel range for the right ear in all three charts is indicated by the line with circles; for the left, by the line with crosses.

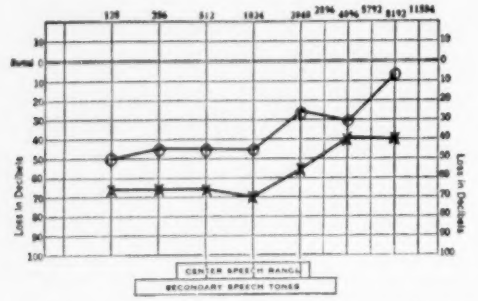

Chart 2.—Audiogram made on Nov. 10, 1948 at the height of an attack.

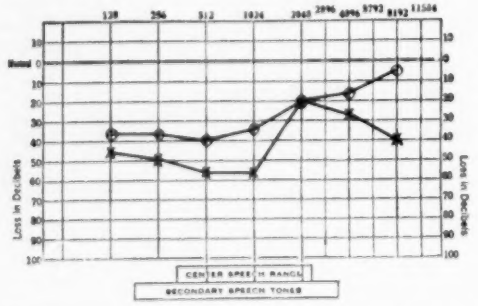

Chart 3.—Audiogram made on November 11, twenty-four hours after treatment with meralluride sodium chloride solution.

Pollens: Sweet vernal grass, red top and corn.

Molds: Candida, Mucor.

Foods: Beet, Brazil nut, chocolate, codfish, corn, crab, lamb, oat, pork and tomato.

The patient was seen during his first attack of Ménière's symptom complex while under my care on Nov. 10, 1948. He stated that since November 6 he had noticed an increasing number of hives and a definite decrease in hearing. On being questioned, he proffered the information that he had eaten large quantities of chocolate for several days. His dizziness was quite severe; he had a tendency to fall to the right and was extremely nauseated. Examination of the ears, nose and throat showed no change. An audiogram demonstrated an increased impairment of hearing (chart 2). The patient's weight was 143 pounds (65 Kg.); the blood pressure was 118 systolic and 76 diastolic, and the pulse rate was 64. He was given 2 cc. of meralluride sodium solution intramuscularly and was asked to avoid salt and to restrict the intake of fluids. He improved greatly the following day, November 11, and was feeling normal except for a slight "hangover sensation." There was a definite increase in hearing (chart 3), and the tinnitus disappeared. The blood pressure and pulse rate were unchanged, but the body weight dropped to 138 pounds (62.6 Kg.), with a urinary output of 3,500 cc. within eighteen hours. He was requested to return for a second injection on November 12, but because of his improvement he failed to keep the appointment.

On November 16 an exacerbation of symptoms occurred. It was revealed that the patient had consumed a large quantity of fluid in fruit form and chocolate ice cream. His weight at this time was 140 pounds (63.5 Kg.). Audiometric examination showed a further impairment in hearing. An injection of 2 cc. of meralluride sodium chloride solution was given, with the same definite improvement being obtained within twelve hours.

This patient has been observed during several subsequent attacks, each following the same pattern, with hives, tinnitus, vertigo and decrease in hearing. The last two attacks have both been preceded by ingestion of unusual amounts of pork and pork products. Each attack has responded so well to injections of meralluride sodium solution that the patient has lost in all only one day from his job as a meat cutter.

Comment.—This patient demonstrated a well defined Ménière's syndrome, associated with urticaria and sensitivity to certain foods. It is believed that the limited amount of nerve deafness may be accounted for by the early stage of the illness. Meralluride sodium solution produced a remarkable improvement on repeated occasions by means of massive dehydration.

A number of drugs, including quinine, salicylates and digitalis, have been described as producing toxic labyrinthitis and damage to the eighth nerve. Dragstedt[34] stated that it is probable that some drugs produce both allergic and nonallergic types of toxic reactions, and he proposed certain criteria, into which fit the sequelae of streptomycin therapy. The incidence of labyrinthine damage as a toxic reaction bears direct relation to the dosage in and duration of streptomycin therapy. How-

34. Dragstedt, C. A.: Idiosyncrasy to Drugs, J. A. M. A. **135**:133-136 (Sept. 20) 1947.

ever, the eosinophilia seen in some cases in which streptomycin therapy was being given has been interpreted as a toxic-allergic phenomenon.[35] Fowler and Seligman[36] cited a case in which otic symptoms occurred on the first day of the second course of treatment with streptomycin and speculated on the possibility of sensitization. It is possible that the untoward action of streptomycin on the auditory and the equilibrium system may in part be due to allergy, with these systems acting as shock organs.

COMMENT

A complete appreciation of allergy is essential to the prophylaxis of aural diseases. Studies which are especially applicable are those concerned with the prevention of deafness, which often follows repeated infections of the poorly resistant allergic mucosa of the nose and ear. Children who manifest allergic conditions or who have a family history of allergy deserve careful observation and therapy. Mao,[37] on the basis of studies conducted at the Pennsylvania School for the Deaf, found a large percentage of cases with biologic or inherited deafness associated with allergy. The recognition of allergy of the upper respiratory tract is necessary in aviation medicine because the presence of allergy prevents proper ventilation of the nose, proper breathing of oxygen and has a tendency to obstruct the ostia of the sinuses and eustachian tubes, thus becoming a major factor in causing aerosinusitis and aero-otitis media.[38]

In the diagnosis of allergy, as well as for its treatment, a thorough history is important. In many cases a diagnosis can be made from the history, which must be complete. The allergic state can also be recognized by physical examination, when characteristic changes of the mucous membrane, including polyps, are present. As an adjunct, routine skin tests are performed. Patch tests are necessary for the diagnosis of contact dermatitis. Scratch and intracutaneous tests are applied alone or in combination in the study of inhalant and food allergy. Other laboratory studies include blood counts for eosinophilia and the leukopenic index, for confirmation of the allergic state and food sensitivity, respectively. Some investigators[39] found that the response to epinephrine, consisting in alleviation of symptoms following subcutaneous injection of the drug, is helpful in the diagnosis of aural allergy. A

35. Arany, L. S.: Streptomycin and Eosinophilia, Correspondence, J. A. M. A. **135**:180 (Sept. 20) 1947.

36. Fowler, E. P., Jr., and Seligman, E.: Otic Complications of Streptomycin Therapy, J. A. M. A. **133**:87-91 (Jan. 11) 1947.

37. Mao, C. Y.: Allergy as a Contributing Factor to Biologic Deafness, Arch. Otolaryng. **35**:582-586 (April) 1942.

38. Poos, E. E.: Allergy of the Upper Respiratory Tract, J. Aviation Med. **15**:264-271 (Aug.) 1944.

39. Feinberg and Durham,[16a] Kuhn.[18a]

cytologic report on nasal smears, particularly that of secretion from the eustachian orifice, should be obtained routinely in cases in which there is suspicion of allergy.

Since it is not the purpose of this paper to discuss therapy, only the essential principles will be enumerated:

1. Specific hyposensitization should be undertaken when feasible.
2. Environmental factors responsible should be removed or avoided.
3. Foods causing sensitivity should be omitted from the diet.
4. Local and general therapy should be instituted when indicated, with use of epinephrine and ephedrine-like compounds or antihistaminic drugs.[40]
5. The program for the detection and treatment of allergy should be performed in conjunction with rhino-otologic therapy. It is important that neither be neglected.

SUMMARY

In summarizing, several basic factors should be emphasized.

Allergic diseases of the ear, as well as those of the nose and throat, are frequently unrecognized.

The only way to diagnose allergy is to suspect allergy and to be especially aware of the patient with a personal or family history of allergy.

Allergy may be responsible for common types of aural disease, such as external otitis, eustachian blockage, disease of the middle ear and Ménière's symptom complex.

Many of the permanent changes caused by allergy may be prevented by early recognition and treatment.

The treatment of allergic conditions of the upper respiratory tract should consist in the simultaneous management of local lesions and allergy. This treatment can be performed best by the otorhinolaryngologist who is well informed in problems in allergy.

1616 Pacific Avenue.

40. Dintenfass, A.: Evaluation of Antihistaminic Drugs, J. M. Soc. New Jersey **46**:66-68 (Feb.) 1949.

ENDAURAL RADICAL MASTOIDECTOMY FOR CHRONIC MASTOIDITIS

R. J. McQUISTON, M.D.
INDIANAPOLIS

THE OTOLOGIST who is consulted by a patient with a chronically discharging ear must first make a study of the pathologic condition present. In some cases of continued aural discharge, the pathologic process may be of no danger to the patient's life; whereas in other cases there is found an extremely dangerous situation awaiting only an acute exacerbation, which may lead to serious intracranial complications and threaten the life of the patient.

The locations of the perforations in the membrana tympani and the character of the discharge are of significance. Perforations in the anterior inferior quadrant, or so-called tubal perforations with an odorless mucoid type of discharge, are, as a rule, due to nasopharyngeal infections and are not considered serious. Central perforations of the drum membrane with a mucoid type of odorless discharge are usually due to mucosal infections confined to the middle ear itself and may not be dangerous. Marginal tympanic perforations, or attic perforations, usually indicate bony necrosis, involving the bony walls of the tympanic cavity, the epitympanum, the adjacent mastoid cells or the ossicles. In these types of perforations cholesteatomas and granulation tissue may be found. The exudate is thin and foul. This picture presents a dangerous type of aural disease regardless of the fact that the patient is otherwise healthy.

So far as the patient is concerned, he is usually interested primarily in a cure for the discharge from his ear. He has long since become accustomed to his loss of hearing, and he is unaware of any dangers which later may threaten his life.

It is the duty of the otologist to evaluate the pathologic condition and to recommend treatment. Audiometric studies should be made to determine if useful hearing is present, and if it is, every effort should be made to preserve this hearing in eradicating the infection.

In some cases in which the condition is confined to the epitympanic region, the patient may have useful hearing, indicating that the ossicular chain is intact and functioning. In such a case the modified radical

From the Department of Otolaryngology, Indiana University School of Medicine.

mastoidectomy, or the atticomastoidectomy, is indicated. However, in our experience, there have been only a limited number of cases that have fallen into this category. Encountered in the majority of epitympanic infections, especially if a cholesteatoma is present, is necrosis of the incus, the incomallear joint or the articulation of the long process of the incus with the stapes. If this condition is present there is no reason to leave the malleus and incus in situ, and if they are allowed to remain they will only act as foreign bodies, resulting in the persistence of the aural discharge after operation.

The radical mastoidectomy which is done to eradicate the infection and to obtain a safe dry ear as an end result must be carefully and painstakingly done. The endaural approach provides adequate exposure and is a direct route to the middle ear and epitympanic regions.

Fig. 1.—The cartilaginous external ear moderately retracted, showing its relation to the underlying mastoid bone. *A* indicates the tragus; *B*, the helix; *C*, the anthelix; *D*, the margins of the conchal cartilage, and *E*, Henle's spine.

It gives the surgeon the best opportunity to remove completely the pathologic material.[1]

The endaural approach in temporal bone surgery and the use of the electrically driven burr has been developed and popularized in this country by Lempert. The endaural incisions used to expose the mastoid process are extracartilaginous.[2]

In the fenestration technic Lempert uses three incisions in removing a triangular piece of skin, whereas in the radical mastoidectomy

1. Morrison, W. W.: Endaural Surgery for Chronic Mastoiditis, Ann. Otol., Rhin. & Laryng. **56**:317-327, 1947.

2. Lempert, J.: Endaural, Antauricular Surgical Approach to the Temporal Bone, Arch. Otolaryng. **27**:555-587 (May) 1938.

he uses four incisions in removing the skin of the posterior canal wall up to the margins of the conchal cartilage.[3] This skin is later used as a skin graft.

Shambaugh uses a single incision, beginning below at the level of the floor of the external meatus along the edges of the conchal cartilage and extending upward between the helix and the tragus.

House [4] has modified these approaches, using three incisions similar to the ones advocated by Lempert. The first incision is made more externally in the skin of the auditory canal; the second incision is made in the usual manner, and the third follows the edges of the conchal cartilage. The triangular area of skin removed is very small.

In radical mastoid surgical procedures, in an effort to secure as much skin as possible for a subsequent skin graft, I have used a technic

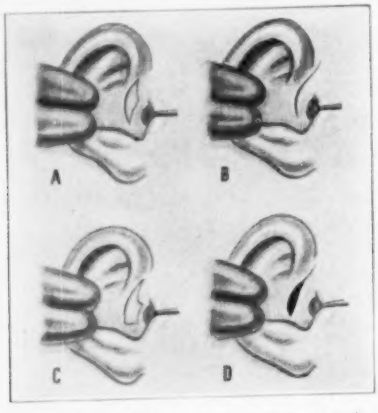

Fig. 2.—Endaural incisions. *A* shows Lempert's fenestration; *B*, Shambaugh's endaural incision; *C*, Lempert's endaural incisions used in radical mastoidectomy, and *D*, House's endaural incisions.

which involves removing all the skin of the auditory canal which does not cover the cartilaginous external ear. One incision beginning at the drum membrane at the anterior superior portion of the bony canal is made upward along the petrotympanic fissure line to the top of the bony canal and then is continued upward between the helix and tragus to the level of the anthelix, becoming more superficial, to avoid the temporal muscle, as the linea temporalis is reached. Beginning anteriorly at the top of the bony canal at the petrotympanic fissure, a second

3. Lempert, J.: Lempert Endaural Subcortical Mastoidotympanectomy for Cure of Chronic Persistent Suppurative Otitis Media, Arch. Otolaryng. **49**:20-35 (Jan.) 1949.

4. House, H. P.: Surgery of the Chronically Discharging Ear, Arch. Otolaryng. **49**:135-150 (Feb.) 1949.

incision is continued downward along the junction of the bony canal and the cartilaginous tragus across the floor of the external meatus and continued upward along the margin of the conchal cartilage joining the first incision above, between the helix and tragus. By subperiosteal elevation, the skin of the auditory canal is entirely removed down to the tympanum. This large piece of skin is preserved in warm saline solution for future use as a skin graft. This endaural technic is, perhaps, more time consuming, because the skin of the anterior wall and floor of the bony canal is very thin, making elevation slightly more difficult. However, because of the thinness, it makes an excellent skin graft—otherwise it would be destroyed and wasted. In order to prevent postoperative stenosis of the external canal, I remove the

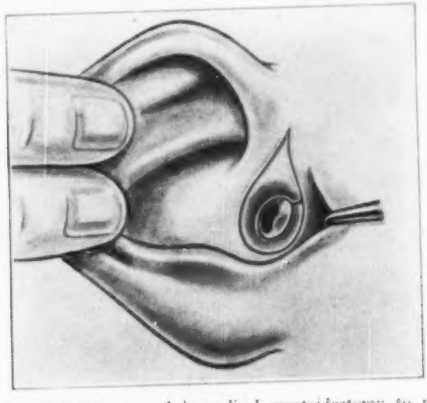

Fig. 3.—Endaural incisions used in radical mastoidectomy to remove all the skin of the external auditory canal which does not cover the cartilaginous external ear. This skin is later used as a skin graft.

periosteum and subcutaneous tissue beneath the conchal cartilage covering the mastoid process and that beneath the temporal muscle.[4]

After retraction, an excellent exposure of the middle ear and mastoid process is obtained. With Henle's spine as a guide and the slant of the bony canal taken into consideration, the location of the mastoid antrum is determined. Postauricularly, one is likely to be guided only by Henle's spine and to lose sight of the slant of the bony canal, which is not directly visualized. This oversight may lead to difficulty in locating the antrum in some temporal bones. By the endaural approach, especially with the soft tissue of the canal wall removed, the mastoid antrum is easily located. Lempert has advocated entering the mastoid antrum directly through the canal wall at the junction of the external two thirds, with the inner third of the bony canal on a line from

Henle's spine to the tympanum.[5] Later, he described a method of entering the epitympanum directly through the notch of Rivinus, after widening the external auditory canal, thus eliminating the search for the mastoid antrum.[5] For those who have had less experience in temporal bone surgery, I have found that the antrum may be safely approached by gradually enlarging the outer three fourths of the bony canal in its posterior and superior portions, beginning just above the tympanum and extending up to the external margins of the bony canal, using a small round, motor-driven cutting burr. This gradual enlargement of the canal is extended superiorly and posteriorly, and soon the periantral cells leading to the antrum make their appearance. This method is a safe and easy way of locating the antrum and is particularly valuable to the surgeon who is learning the endaural technic. After

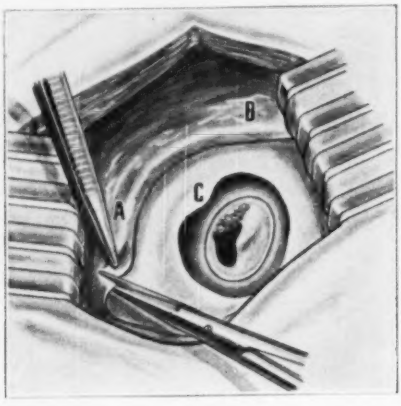

Fig. 4.—Retraction of the superficial soft tissue. *A* indicates the periosteum and subcutaneous tissue beneath the conchal cartilage; *B*, these tissues also removed superiorly beneath the temporal muscle to prevent postoperative stenosis of the external auditory canal, and *C*, Henle's spine.

the antrum is located and the periantral cells are removed, the horizontal semicircular canal is readily identified. By this procedure the surgeon is orientated early in the operation and is prepared to continue his work with an excellent exposure of the operative field. The radical mastoidectomy is primarily an operation on the middle ear; whereas the pathologic condition in the mastoid process, in many cases, is minimal. In cases of long-standing infections, particularly those originating in childhood and carrying over into adult life, the mastoid process becomes extremely sclerotic and remains small, owing to its lack of

5. Lempert, J.: Complete Endaural Mastoidectomy, Monatschr. f. Ohrenh. **64**:143-159, 1930.

development—probably due to an early infection. The body tends to carry on a healing process, first by decalcification, then by sclerosis. In this type of case, most of the pathologic changes in the mastoid process itself are in the antrum and adjacent periantral cells. This diseased matter is removed, exposing the middle fossa plate above and posteriorly removing only as much of the sclerotic bone as is necessary to exteriorize completely the underlying diseased area. The horizontal canal is delineated beginning superiorly and skeletonized to its posterior extension, and the posterior semicircular canal is located. In temporal bone surgery, after the antrum is located the horizontal canal is the most important immediate landmark. It is the key to the location of the other vital structures to be encountered and a guide to the mastoid course of the facial nerve, which starts its descent through the

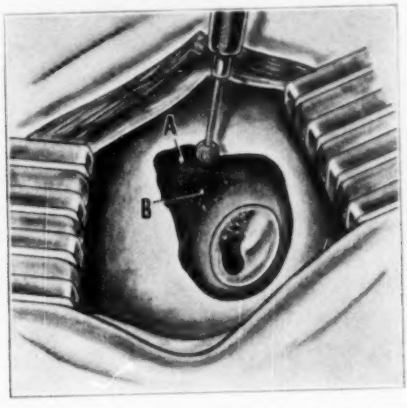

Fig. 5.—The periantral cells leading to the mastoid antrum exposed by gradually enlarging the external three fourths of the posterosuperior portion of the external bony canal. *A* indicates the mastoid cavity, *B*, the periantral cells leading to the mastoid antrum.

mastoid process, at a slightly deeper level, at about the posterior extension of this canal, and continues its course straight down to the stylomastoid foramen, which is at the level where the digastric ridge joins the posterior bony canal wall, where it leaves the mastoid and passes into the neck.

In approaching the mastoid antrum by enlarging the external three fourths of the bony canal wall in its posterosuperior portion and gradually entering into it, one now has left only a small amount of bone forming the bridge between the antrum and the middle ear. This bridge of bone is thinned down by the burr and is removed with a small rongeur before the incus, malleus or contents of the middle ear are disturbed. The incus lies over the horizontal canal and facial

nerve in the incudal fossa and articulates with the head of the malleus anteriorly, while its long process articulates with the stapes internally. In its position it protects the facial nerve and the stapes from possible injury while this last bridge of bone is being removed. After removal of this bridge the notch of Rivinus is further widened by removing its anterior and posterior spurs flush with the canal wall anteriorly and with the facial ridge posteriorly, which is taken down to the level of the horizontal canal. After this procedure is carried out, the incus can easily be teased loose from its articulations with the head of the malleus and the stapes and removed. After removal of the incus the facial nerve and stapes can plainly be visualized.

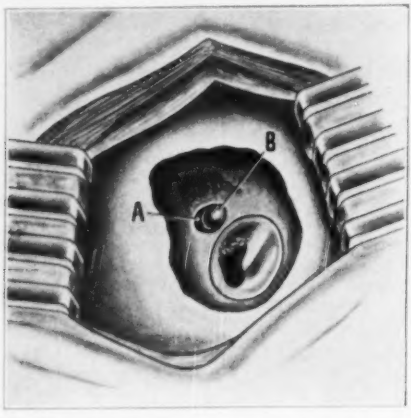

Fig. 6.—*A*, mastoid antrum opened after removal of the periantral cells; *B*, horizontal semicircular canal.

At this point the operation has really just begun, for the seat of the infection, which prompted the patient to consult the surgeon, has been reached. Unfortunately, in the past when the surgeon reached this point, he made a few more daring swipes with the curet, hoping to avoid the facial nerve and not to dislocate the stapes, and he was ready to quit. This was unfortunate for the patient, because the chronic discharge of the ear would probably still persist, and unfortunate for the surgeon also, because he had robbed himself of the most beautiful and gratifying part of the operation.

With the use of magnification at this stage of the operation, the detailed anatomy of the middle ear can be visualized on removal of the infected debris. At this point the middle fossa plate is further exposed anteriorly into the attic of the middle ear and the superior canal identified. With the operator continuing from above, and anterior

to the horizontal canal, the infected tissues, consisting of cholesteatomas, granulations, remnants of the middle ear ossicles or membrana tympani, are gently elevated downward over the horizontal canal and the facial nerve. The cochleariform process of the facial canal and the bony groove with the tensor tympani muscle are now seen. This cochleariform process is removed and the tensor tympani muscle elevated out of the bony groove and removed from its origin in the eustachian tube along with the peritubal cells. The cells in the hypotympanum are exenterated, and the floor of the external bony canal wall is lowered to the level of the hypotympanum to prevent a pocketing of infected materials in this area. The posterior canal wall is further lowered, and the pyramidal process, which forms an overhang over the stapes in the oval window, and the niche of the round window

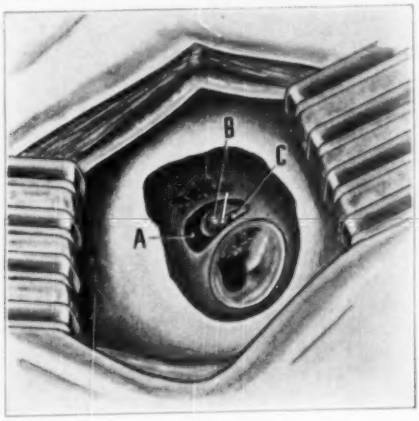

Fig. 7.—*A*, mastoid antrum enlarged and the bridge between the middle ear and the mastoid antrum thinned down; *B*, horizontal semicircular canal, and *C*, incus.

are removed, bringing these structures, as well as further posterior extension of the facial nerve, into view. In the floor of the middle ear, anterior and inferior to the promontory and deep to the eustachian tube, is the carotid canal; posterior to the promontory in the hypotympanum, at a lower level, is the jugular bulb; involved cells in these regions are removed by curettement. Further minute dissection is needed to remove completely the infected granulations or cholesteatomatous material from the regions of the stapes and oval window. This removal is usually carried out with a small ring curet and a paracentesis knife, with these instruments working in the direction from posterior to anterior above and below the stapes and the stapedius tendon, parallel to the facial canal. This technic minimizes the danger to the

facial nerve and reduces the chances of dislocating the stapes, for the pull of the stapedius tendon tends to prevent tilting and dislocating of this ossicle from the oval window, as described by House.[4] Granulations involving the round window are also removed by minute dissection. After the osseous pathologic material has been thoroughly removed, the skin which had been removed from the external canal is trimmed and thinned out and replaced carefully in the middle ear, anterior lipping entirely covering the orifice of the eustachian tube. I use the thickest portion of the skin graft to cover this area, with the thinner portions covering the stapedial area, attic, facial ridge and adjacent mastoid floor. The graft is held in position with pledgets of paraffin-gauze mesh packing. Two black silk sutures are used to close the skin incision at its upper portion. In some cases in which

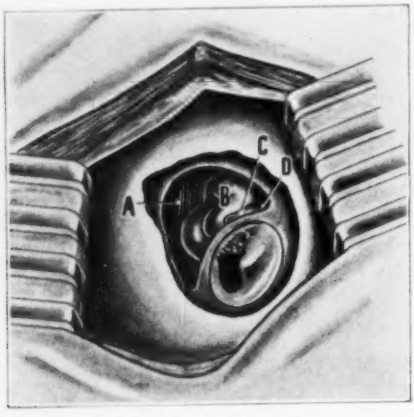

Fig. 8.—The semicircular canals defined and the dissection carried forward into the attic of the middle ear. *A* indicates mastoid cavity; *B*, labyrinth; *C*, incus, and *D*, head of the malleus.

there is a rather large cavity to epithelize or in cases of reoperation on radical mastoidectomy cavities in which the skin of the canal is not usable I use skin from the thigh and line the entire cavity. These grafts should be practically full thickness—thin grafts will curl up and are difficult to handle. I have been using the newly devised Brown "electrodermatome" for taking the skin from the thigh. This instrument is a rather ingenious one, which is easy to operate and takes the skin evenly at any desired thickness.

As for the immediate postoperative care, penicillin is given—300,000 units daily for three days—and the patient is allowed to be up on the second or third postoperative day. The packing is removed on the fifth postoperative day and no further packing is used during the

patient's stay in the hospital. The large dressing and the sutures are also removed at this time, and the patient is allowed to go home on the sixth or seventh postoperative day.

The application of the skin graft, which covers the floor of the middle ear, lipping anteriorly to cover the orifice of the eustachian tube and lining the attic above and the floor of the exposed mastoid posteriorly, has been practiced by Lempert for several years. I have applied this technic for the last four years, and I have been very happy with the results. However, the success of any skin grafting in the mastoid and middle ear cavity depends on how thoroughly and painstakingly the mastoid surgery is done. Any areas of cholesteatomas, granulations or necrotic bone that remain will spell failure of the

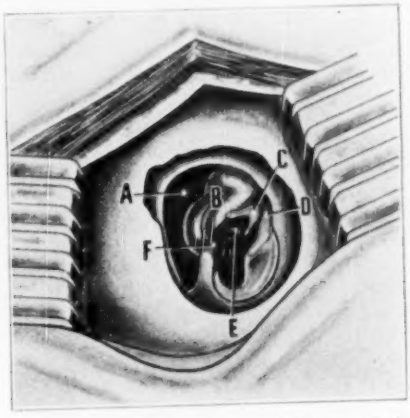

Fig. 9.—The bridge of bone between the middle ear and the mastoid cavity removed before the incus, malleus or contents of the middle ear are disturbed. *A* indicates mastoid cavity; *B*, labyrinth; *C*, incus; *D*, head of the malleus; *E*, stapes, and *F*, facial ridge and pyramidal process.

skin graft, as well as failure to cure the patient of the chronic discharge of the ear.

As is well known, the postoperative care of the radical mastoidectomy cavity is one that requires time and patience. I am sure that all otologists do not get completely dry, epithelized ears within two months, with or without the use of skin grafts. We otologists have all seen ears that we think we have operated on well, and at the completion of the operation have breathed a sigh of satisfaction at the fine, painstaking job we have done, only to see the ear proliferate mountains of granulations and persistent purulent discharge. Many will say the patient has an infected cavity, but I think that not even an ear most perfectly operated on has been rendered germ free. When confronted with these problems in an ear already operated on, I prefer to remove

these granulations by curettement throughout the entire cavity up to the edges of the skin graft or epithelized area and then to repack it with petrolatum packing. This procedure may have to be repeated several times, and may grow extremely tiresome to both the patient and the physician, but in my hands it has proved the most successful method of treatment. I no longer use caustics, such as silver nitrate, to subdue these granulations.

Postoperative stenosis of the external auditory canal occasionally occurs after endaural mastoid surgery and may require further removal of some of the soft tissue to restore the desired opening. This restoration is accomplished by removing some of the tissue along the margin of the conchal cartilage posteriorly and also removing a V-shaped wedge from between the tragus and helix above. The complication

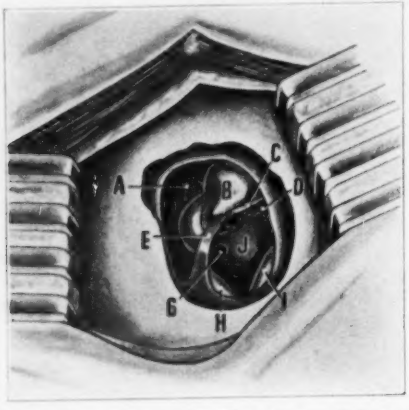

Fig. 10.—The completed radical mastoid cavity. *A* indicates mastoid cavity; *B*, labyrinth; *C*, facial nerve; *D*, location of the tensor tympani muscle and cochleariform process, which have been removed; *E*, facial ridge; *G*, round windows; *H*, jugular bulb; *I*, carotid canal, and *J*, promontory of the cochlea.

of postoperative stenosis may be prevented in many cases by removing this wedge of subcutaneous tissue beneath the temporal muscle at the time of the operation, as described by House. Prevention of postoperative stenosis is also accomplished by the incision I have been using together with removal of the subcutaneous tissue beneath the conchal cartilage over the mastoid process. Stenosis of the canal probably occurs more frequently in the postauricular type of radical mastoidectomy, which, in itself, is not purely postauricular, for it also includes a plastic repair of the external canal, which makes it a combination of an endaural and a postauricular operation.

The type of operation described here is possible only if the surgeon has trained himself to identify all the vital structures of the middle

ear under magnification. If these structures are plainly visualized and identified, the danger of injuring them is minimal. Those who have been trained in the fenestration technic will agree that one does not just equip oneself with a drill, a magnifying loop and an electric headlight and begin operating on patients. Changes and refinements in one's operating technic must be made while working with

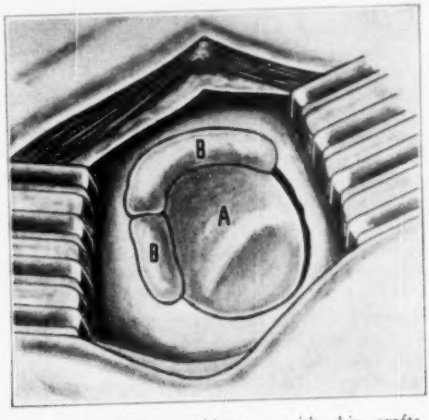

Fig. 11.—Completed radical mastoidectomy with skin grafts in position. *A* indicates skin graft from the external auditory canal and *B*, additional skin grafts from the thigh, if needed.

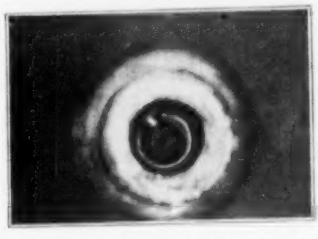

Fig. 12.—Healed epithelized radical mastoid cavity.

the field under magnification, which requires considerable practice on the cadaver to get accustomed to using the drill and to become familiar with the vital structures encountered. The development of the fenestration technic by Lempert and others and its application to the radical mastoidectomy and other types of otologic surgery represent one of the outstanding contributions to this field.

608 Guaranty Building.

Case Reports

EXTRAMEDULLARY PLASMA CELL TUMOR

FREDERICK FUERSTE Jr., M.D.
MILWAUKEE
AND
SAM S. ZUCKERMAN, M.D.
CHEYENNE, WYO.

This is a case report of extramedullary plasma cell tumors occurring in the nasopharynx, the duodenum and in multiple sites on the skin of a man aged 56. Mattick and Thibaudeau [1] deemed all such interesting and rare tumors worthy of reporting, and the multiplicity of the extramedullary sites supplements particularly the interest in this case.

The first case of an extramedullary neoplastic growth simulating that seen in bone lesions of multiple myeloma was described by Schridde in 1905. Since that time there have been about 150 cases described in the literature under the terminology of extramedullary plasma cell tumor, extramedullary plasmoma, plasmocytoma and plasma cell sarcoma. The most comprehensive review of the literature is that by Hellwig [2] (1943), covering 127 cases of others and 1 of his own; in 65, the tumor originated in the upper respiratory tract; in 50, in the conjunctiva—their benignity suggesting inflammation rather than neoplasm; in 4, in the lymph nodes, and in 13 in areas outside these sites—the pleura, mediastinum, thyroid gland, ovary, intestines and urogenital organs. In 30 of the 65 cases of involvement of the respiratory tract the growth was "definitely invasive" or "cancerous." Grossly, these lesions of the upper respiratory tract were soft, friable, gray or yellow growths, with smooth, cauliflower or ulcerated surfaces, often necrotic. Symptoms were first produced mechanically, then were manifest by invasion of nerves, hemorrhages, foul discharge, swelling of the nose and cheeks, encroachment on the palpebral fissures and lacrimation. Bence Jones proteinuria was absent as long as bone was uninvolved.

Rosenwasser's [3] case of a plasmocytoma of the nasal cavity in which extensive irradiation therapy was given is interesting to note. Twelve months after the last treatment and three years after the beginning of therapy, the patient died of carcinoma of the stomach with widespread metastasis, but no evidence of residua at the primary site could be found.

Of subsequent reports, Haines [4] (1942) described a single case of metastasizing tonsillar plasma cell tumor; McNamara and Rogers [5] (1943) reported on a single

1. Mattick, W. L., and Thibaudeau, A. A.: Extramedullary Plasma Cell Tumors of Upper Air Passages, Am. J. Cancer **23**:513, 1935.

2. Hellwig, A.: Extramedullary Plasma Cell Tumors as Observed in Various Locations, Arch. Path. **36**:95 (July) 1943.

3. Rosenwasser, H.: Plasmocytoma of the Nasal Cavity, Laryngoscope **40**:576, (Aug.) 1930; Further Observations on Case of Plasmocytoma of Nasal Cavity: Case of Nasal Plasmocytoma and Carcinoma of Stomach, ibid. **44**:826 (Oct.) 1934.

4. Haines, M.: A Metastasizing Plasma Cell Tumor of the Pharyngeal Tonsil, J. Laryng. & Otol. **57**:264, 1942.

5. McNamara, W. L., and Rogers, R. J.: Extramedullary Plasma Cell Tumor of a Tonsil with Metastasis, Arch. Path. **36**:89 (July) 1943.

plasma cell tumor of a tonsil with a radiosensitive cervical metastasis; Gordon and Walker [6] (1944) reported the removal by lobectomy of a plasma cell tumor from the lung, and Stancil and Tomlinson [7] (1944) reported success with roentgen therapy on an ulcerating destructive plasma cell growth of the palate and pillars in a 26 year old Salvadorian.

Figi, Broders, and Havens [8] (1945) reported a series of 11 cases of plasma cell tumors of the upper respiratory tract observed over a fourteen year period (1930-1943), in 6 of which the growth took origin in the maxillary sinuses. All these tumors seemed highly malignant, roentgenologic evidence revealing more destruction than gross physical examination suggested. One patient with a tonsillar plasma cell tumor returned for examination, eight years after surgical and roentgenologic therapy, with multiple metastasis to the bone and a Bence Jones proteinuria, both conditions not being present on the patient's previous admission to the hospital. In 1946 Connor [9] reported the removal of a benign plasmocytoma from the soft palate of a 57 year old man.

Concerning plasmocytoma involving the intestinal tract, Brown and Liber [10] in 1939 reported the case of a Negro aged 57 whose ileum was infiltrated with masses of yellowish white firm tissue, revealing solid sheets of invasive plasma cells with a few lymphocytes. This report included cases of other authors: (1) Vasiliu and Popa (findings at autopsy in a 32 year old woman with ulcerating nodular plasma cell tumors in mucosa of the stomach, ileum and sigmoid colon); (2) North (30 cm. of resected ileum for obstruction, showing infiltration of all bowel layers by plasma cells); (3) Razzaboni (multiple plasma cell tumors of terminal ileum, colon and appendix), and (4) Vallone (resected ileum for obstruction, occluded by plasma cell tumor).

In 1945 Esposito and Stout [11] reported additional cases of lesions of the intestinal tract, one report being on a 35 year old Costa Rican with annular soft constrictions of plasmocytomas of the jejunum and metastasis to the regional lymph nodes. Couret [12] in 1946 described the involvement of the muscular coat of the stomach in a 48 year old white woman by infiltrating plasma cells and lymphocytes. In 1947 Schwander, Estes and Cooper [13] reported a case of plasmocytoma of the stomach. Arel [14] (1946) contributed 3 cases from Turkey: 1 of obstructive plasmoma of the

6. Gordon, J., and Walker, G.: Plasmocytoma of the Lung, Arch. Path. **37**:222 (March) 1944.

7. Stancil, J. R., and Tomlinson, W. J.: Extramedullary Plasma Cell Tumor of the Mouth, Arch. Otolaryng. **40**:139 (Aug.) 1944.

8. Figi, F. A.; Broders, A. C., and Havens, F. Z.: Plasma Cell Tumors of the Upper Part of the Respiratory Tract, Ann. Otol., Rhin. & Laryng. **54**:283 (June) 1945.

9. Connor, C. E.: Solitary Extramedullary Plasmocytoma: Case Report, Ann. Otol., Rhin. & Laryng. **55**:342 (June) 1946.

10. Brown, C. R., and Liber, A. F.: Multiple Plasmoma of the Ileum and Colon, Arch. Path. **28**:112 (July) 1939.

11. Esposito, J. J., and Stout, A. P.: Multiple Plasmocytoma of the Jejunum; Report of a Case, Am. J. Roentgenol. **53**:33 (Jan.) 1945.

12. Couret, J. S.: Extramedullary Plasma Cell Tumor of the Stomach: A Case Report, Am. J. Clin. Path. **16**:213 (March) 1946.

13. Schwander, H.; Estes, J., and Cooper, W. G.: Plasmocytoma of the Stomach: Report of a Case, Am. J. Path. **23**:237 (March) 1947.

14. Arel, F.: Contribution to the Study of Extramedullary Plasmocytoma, J. Internat. Coll. Surgeons **9**:225 (March-April) 1946.

distal ileum in an 8 year old boy with no metastasis; 1 with involvement of the cecum and appendix and postoperative recurrence in a 15 year old boy, and 1 of a plasmocytoma of the nasal mucosa in a 35 year old man. Hellwig included the reports of 2 cases of plasmocytoma of the skin.

REPORT OF A CASE

A white man aged 56 sustained an exceedingly severe, depressed skull fracture in 1935 from a road blast, which left him with complete hemiplegia on the right, hemianesthesia, facial paralysis and anesthesia on the left, complete motor aphasia—verbal and nominal—agraphia and alexia. In the subsequent four to five months, his strength and ability to perceive sensations gradually returned. During the following four years his wife taught him to speak, to say a few words and sentences and to identify simple written letters and numbers. Approximately one year after the accident the patient began having typical grand mal seizures twice a week, which diminished in frequency until 1945, after which he was free from such attacks.

Of significance is the patient's statement that prior to his accident he had had no trouble with digestion, but approximately one year after the accident he began to have seasonal and periodical epigastric pains of moderate severity several hours postprandially, relieved by milk and sodium bicarbonate. There was no history of hematemesis, melena or other complications.

Our records of the patient's first hospitalization were for January 1944, at which time his complaints on entry to the hospital were of midepigastric pain, relieved by milk and sodium bicarbonate, and of the residuals of his head injury. Examination disclosed a parietal osseous defect on the left, no abnormalities of the abdomen and nothing unusual on roentgenologic study of the gastrointestinal tract and gallbladder. Laboratory observations indicated nothing significant. His epileptiform seizures, aphasia and partial hemiplegia on the right were interpreted as being secondary to the old depressed skull fracture on the right, but no organic pathologic condition could definitely be linked to the abdominal complaints at this time.

Two years later, on February 1946, he returned and presented a history of a cold in the head for two months, with nasal obstruction on the left and swelling over the left cheek. A private physician had taken tissue from a growth in the left nostril and informed him that it was "carcinomatous tissue." Examinaiton revealed an obstructive tumor on the lateral wall of the left nasal passage and swelling over the left nostril and left side of the face. He was examined in June at another hospital, where a biopsy specimen was taken; a diagnosis of grade IV undifferentiated carcinoma of the left maxillary antrum was made, and he received a course of radiation therapy.

In October, the patient returned with an interval history of loss of weight (15 pounds [6.8 Kg.]), anorexia, complete nasal obstruction and dull epigastric pains several hours postprandially, relieved by milk, soft boiled eggs and soft food. Our physical examination at this time disclosed that the left nasolacrimal duct was plugged and bulging over the left inner canthus and the left nasal passage blocked with soft friable material, which was also visible through the posterior choana (fig. 1*A*). The abdomen was again normal to palpation. A roentgenogram showed the left maxillary bone, particularly its medial and orbital walls, partially destroyed by tumor. In two months the growth extended over the left frontal sinus. A series of roentgenograms of the gastrointestinal tract was then reported as showing a constant distorted pattern of a crater on the inferior border of the duodenum,

suggestive of an ulcer with no gastric retention. Roentgenograms of the skull showed the old triangular parietal defect on the left and progression of the erosion to bilateral involvement of the ethmoid sinuses by a tumorous growth. A roentgenogram of the chest did not indicate metastasis of the tumor. Clinical laboratory

Fig. 1.—Photographs showing progressive growth of plasma cell tumor.

findings were again within normal limits except that the sedimentation rate was 26 mm. per hour and a gastric analysis showed total acidity of 80 degrees and free acidity of 61 degrees.

Approximately two months later, the growth began to protrude from the right medial canthus, having eroded through the nasal structures. This growth gradually

enlarged; it was soft, red, friable tissue which, after reaching the size of a hen's egg, became necrotic, the surface sloughing to form a foul-smelling, hemorrhagic mass (fig. 1 *B*). The patient manifested a progressive cachexia and complained constantly of pain in the pit of his stomach, for which Sippy diets, antispasmodics, amphojel® (a colloidal suspension of aluminum hydroxide) and a protolysate diet afforded but little relief. There were no symptoms of obstruction. Narcotics were given for relief of pain in the stomach and head. The growths on the head enlarged in size,

Fig. 2.—Photograph showing excised tumor.

encroaching on both palpebral fissures until vision was completely occluded by local extension and retrobulbar pressure (see figure 1*C* made in June 1947). A node the size of a walnut appeared in the left submaxillary triangle, and the lesions of the head grew continuously, forming distorted masses of hemorrhagic, necrotic, foul-smelling neoplasm in grapefruit-size masses. Extensions deformed the soft palate completely. There was progressive anemia. Roentgenograms of the chest still did not indicate metastasis, but terminally pneumonitis developed with moderate leukocytosis.

A few weeks before the patient's death, numerous small cutaneous nodules developed over the shoulders and thighs. These were thought to be the local reaction from hypodermic medications. One week before death severe abdominal pain suddenly developed with moderate rigidity, at which time it was believed that the ulcer had perforated. The pain and rigidity disappeared in two days. The patient died one week later on September 9.

Autopsy Report (twelve hours post mortem).—The face was distorted as described and illustrated (fig. 1C).

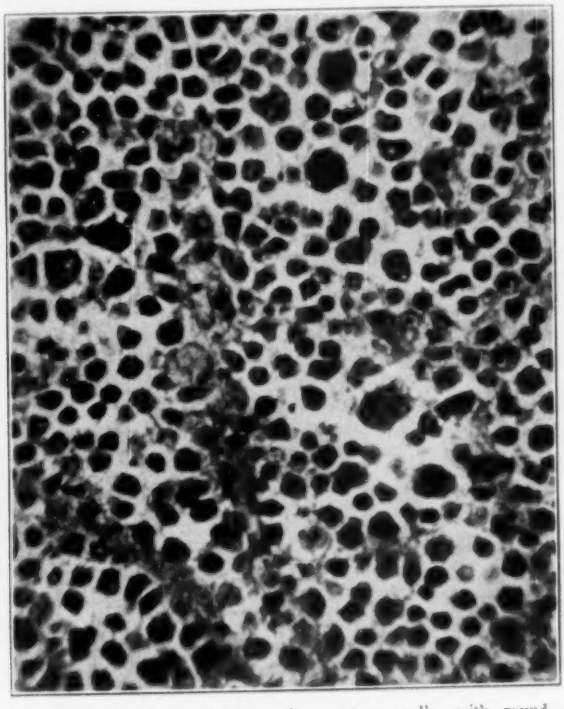

Fig. 3.—Photomicrograph showing plasmocytoma cells, with round, darkly staining nuclei, from duodenal lesion.

The scalp was depressed over a 5 by 8 cm. area of the left temporal and parietal bone to a depth of 1 cm., the skin and dura being the only covering at this point. Over the anterior central gyrus and precentral sulcus of the left cerebral lobe was a 3 by 4 cm. depression in the brain, with fibrous adhesions to the meninges, beneath which was cystic encephalomalacia. The roofs of both frontal sinuses and the sphenoid cells were eroded by the tumor, which had extended into the anterior fossa to elevate the dura and indent the inferior portions of the frontal lobes.

In the skin over the arms, thorax and thighs were numerous shotty nonadherent nodules up to 1.5 cm. in diameter. On section these nodules were grayish, firm and homogeneous.

The lungs were well pneumatized and crepitant except in their dependent portions, which revealed focal consolidation.

The esophagus and stomach were normal. The entire first and second portions of the duodenum were replaced by a firm mass completely encircling the duodenum for a distance of approximately 10 cm. (fig. 2). The third portion of the duodenum and remainder of the intestinal tract were not grossly involved, nor were any

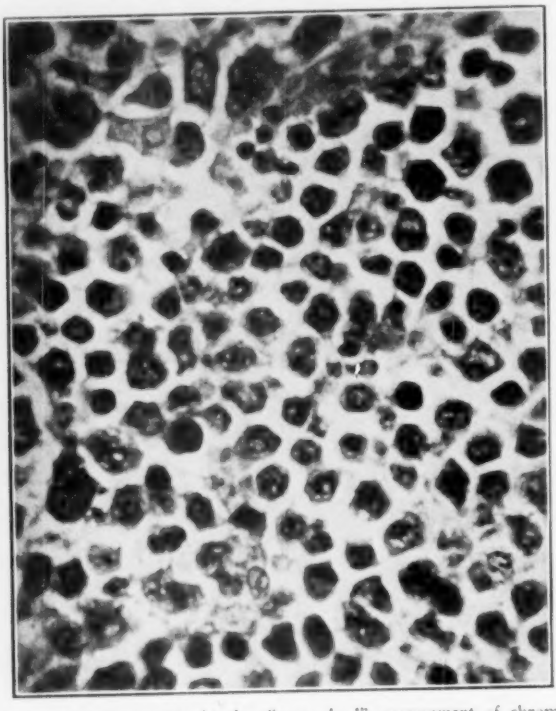

Fig. 4.—Photomicrograph showing "cart wheel" arrangement of chromatin of plasmocytoma cells.

regional or adjacent lymph nodes involved. No crater as shown on the roentgenogram could be identified.

Microscopic Examination.—Microscopic examination of the skin nodules, tumors of the face and duodenum revealed identical tumor cells (figs. 3 and 4). These cells were in nests and masses lying near the vascular network of the connective tissue septums. The cells were large, with large, round, darkly staining nuclei showing a "cartwheel" arrangement of chromatin. Anaplasia and pleomorphism were pronounced. Many of the cells were in mitosis, and some contained giant nuclei.

COMMENT

Opinion on the origin of plasma cell tumor is controversial, as is that of its relation to multiple myeloma and classification as a true neoplasm. Because of Maximow's assertion that plasma cell tumor originates through lymphocytic differentiation, Hellwig suggested that such a tumor may be a lymphoma.

Following are the criteria for identification of plasma cells, as furnished by Marschalko: (1) round, oval or polygonal cells, with abundant nongranular and basophilic cytoplasm without specific granuloplasm; (2) eccentrically placed nuclei; (3) paranuclear light-stained vacuoles or pallor, and (4) small nucleus with from five to eight distinct deeply stained blocks of chromatin masses regularly arranged in a circle about the nuclear membrane forming the "cartwheel nucleus."

Difficulty arises in differentiating plasma cell tumor from chronic inflammatory processes. Hellwig observed that there seemed to be no correlation between the cytologic appearance, the relative maturity of the cells and the clinical behavior or destructive potentiality. In spite of cell structure and mitotic figures, the neoplasm may range from an entirely "noncancerous" growth to a highly invasive one—the difference depending on whether or not the original tumor was localized and noninvasive, i. e., as long as it was localized and confined to soft tissues, a clinical cure was obtainable by surgical excision, adequate irradiation or a combination of both; if the tumor was invasive, no therapy was of more than palliative value. In classifying this tumor among the malignant lymphomata, Jackson, Parker and Bethea [15] stated earlier that there was "no line of demarcation between localized, benign plasmocytoma . . . , and the malignant, fatal multiple myelomata . . . . No clear distinction . . . between the nodular, circumscribed form and the diffuse form." Figi, Broders and Havens stated that all such tumors appeared malignant, varying in degree of malignancy from mild to fulminating. To them these growths in no way suggested simple benign granulomata of "inflammatory nature."

SUMMARY

A fatal case is presented of malignant multiple extramedullary plasma cell tumor, involving the nasopharynx, the duodenum and multiple cutaneous sites. This case suggests the possible etiologic relation of the growth to inflammation or injury at the site of a long-standing duodenal ulcer and sites of hypodermic injections. Supposedly adequate radiation therapy failed to decrease the spread of growth of the nasopharyngeal tumor.

Hynds Building, Cheyenne, Wyo.

15. Jackson, H. Jr.; Parker, F., Jr., and Bethea, J. M.: Studies of Diseases of Lymphoid and Myeloid Tissues: Plasmatocytomata and Their Relation to Multiple Myelomata, Am. J. M. Sc. **181**:169 (Feb.) 1931.

FRACTURE OF STOMACH MAGNET

An Unusual and Interesting Case of Fracture of Magnet in Attempted Removal of Foreign Body from Stomach

MORRIS S. BENDER, M.D.
Director, Department of Otolaryngology, Queens General Hospital
AND
SAM CLAYTON, M.D., D.D.S.
Assistant Attending, Department of Otolaryngology, Queens General Hospital
JAMAICA, N. Y.

Several years ago the General Electric Company introduced a magnet made from an alloy of aluminum, nickel, cobalt and iron, fused under high pressure at almost melting point, in reducing gas. It is the most powerful of several such alloys and is known as "cast alnico-V" and can lift twenty-five times its own weight. Another characteristic is that it can be easily remagnetized should it lose this property. However, this alloy is quite brittle and therefore subject to easy fracture, which may lead to embarrassing complications.

The surgical removal of obstructing metallic foreign bodies in the stomach and duodenum is fraught with danger, even in these days of chemotherapy. Recently, there have been several reports by Murdoch Equen of the use of the "alnico" magnet for extracting metallic foreign bodies from the stomach and the duodenum. By attaching a rod of "alnico" alloy measuring 5 by 0.5 cm. in diameter to an ordinary stomach tube, he was able to extract ferrous foreign bodies from the stomach and duodenum. So simple and innocuous a procedure is a welcome adjunct to the armamentarium in dealing with metallic objects in the stomach and duodenum.

It has been advocated that a similar magnet be utilized for extracting ferrous foreign bodies from the bronchi also. In view of a recent experience of ours, we feel that a word of caution is in order. Were a similar experience to occur in the second and third divisions of the bronchi, it might not end so happily.

REPORT OF CASE

On Sept. 30, 1947, a female Negro child aged 9 months was admitted to the hospital because of an open safety pin in the stomach (fig. 1*A*). The child had swallowed an open safety pin while the mother was changing its diaper. The mother was not certain what type of metal the safety pin was made of. A roentgenogram was taken and revealed an open safety pin located within the fundus of the stomach, lying in a horizontal position with the open end directed toward the pars pylorica.

In view of the possibility that the safety pin may have been of a ferrous material, we felt that this was an ideal opportunity to utilize the stomach magnet that we had at Queens General Hospital. Under fluoroscopic guidance, the magnet end was inserted into the pharynx. The child gulped, and the operator immediately felt a loose piece of metal, which he vainly attempted to grasp. It was then discovered that the magnet had fractured (fig. 2*A*). Under fluoroscopy, the fractured end was easily visible at the cardia (fig. 1*B*). No further manipulation was attempted. The child was placed on a bulky diet, and the progress of the open safety pin and

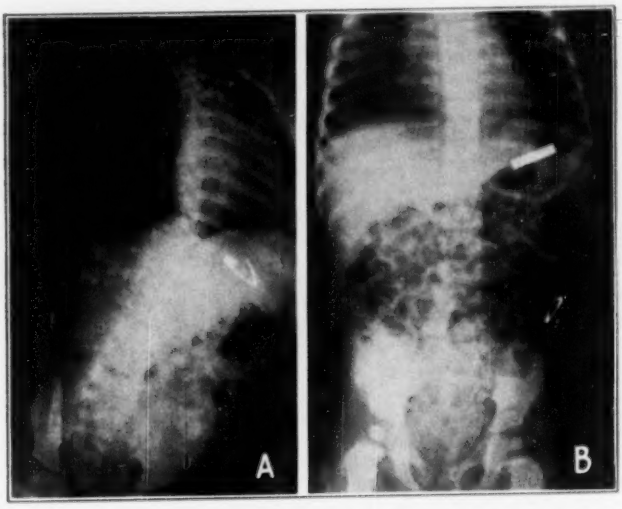

Fig. 1.—*A*, roentgenogram of child's stomach with open safety pin and, *B*, loose fragment of magnet in child's stomach.

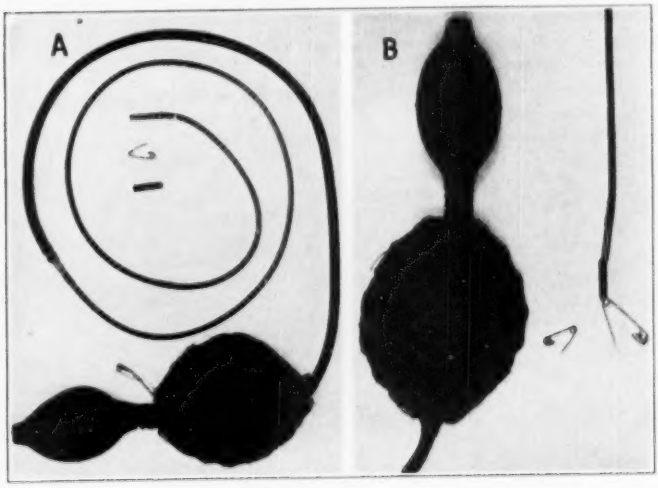

Fig. 2.—*A*, fractured fragment of magnet; *B*, original magnet.

magnet fragment was daily observed by fluoroscopy. On October 2, the open pin and magnet fragment were both passed in the morning bowel movement. The child, having experienced no apparent ill effects whatsoever, was discharged the following day.

COMMENT

Several features of this case are of unusual interest. First, the magnet measured 5 cm. in length by 0.5 cm. in diameter. The loose fragment measured 2.3 cm. and when recovered was found to possess as much magnetism as the original magnet (fig. 2*B*). The portion attached to the stomach tube was found to possess a slight amount of magnetism immediately after fracture. But this property was soon lost. It is a well established fact that the longer a rod, the more susceptible it is to fracture. Since the loose fragment of 2.3 cm. retained the original magnetism, we believe that a magnet of that length would be a safer instrument. If possible, a less brittle magnet should be sought, for if a magnet were to fracture in the segmental bronchi its extraction would become a complicated operation.

Second, the possibility arises that the broken fragment might have been removed at the time of accident because of the retained magnetism in the loose fragment. This possibility could not be determined until the loose fragment had been recovered, and it was then discovered that the entire magnetic property was herein contained.

Third, the open safety pin was found to be made of brass and was not susceptible to the magnet. In view of the fact that safety pins will continue to be the principal means of holding diapers in place, it is suggested that manufacturers of safety pins be advised to use ferrous materials.

SUMMARY AND CONCLUSIONS

A case is presented in which attempted passage of a stomach magnet resulted in fracture of the magnet.

The loose fragment was found to retain as much magnetic property as the previously whole magnet.

Since the metal is so brittle, it is suggested that the magnet portion be made about 2.0 cm. long.

A plea is made for the use of ferrous material in the manufacture of safety pins.

News and Comment

ANNUAL MEETING OF THE AUSTRIAN OTOLARYNGOLOGICAL SOCIETY

The Austrian Otolaryngological Society of Vienna will hold its annual meeting on Sept. 18-20, 1950, in Bad Gastein, Austria. The leading topic will be "Professional Diseases of Ear, Nose and Throat." Further information may be secured from the president, Prof. Dr. E. Schlander, or the secretary, Doz. Dr. O. Novotny, both at Vienna IX., Alserstrasse 4, Austria.

GENERAL NEWS

International Course in Audiology.—A practical course in audiology has been arranged to be held in Stockholm, Sweden, Sept. 11 to 20, 1950. Not all details of the program to be offered are complete at the present time, but following is a tentative list of the subjects. The main emphasis will be placed on practical teaching, and the course is planned to enable every otologist to follow the lectures and demonstrations without any special advance preparation.

A. *Lectures*

I. Anatomic, Physiologic, Physical and Psychologic Background
   1. Anatomy—normal and pathologic
   2. Functional anatomy—physiologic and pathologic
   3. Acoustics and electroacoustics
   4. Psychophysics of hearing

II. Hearing Loss
   1. Prophylaxis
   2. Incidence
   3. Causes (especially traumatic)
   4. Psychology

III. Hearing Tests
   1. Physical basis of audiometry
   2. Comparison between old and modern methods
   3. Tone audiometry—basic and special tests—interpretation
   4. Speech audiometry—basic and special tests—interpretation
   5. Group audiometry—basic and special tests—interpretation
   6. Standardization of determinations, audiometers and audiograms

IV. Treatment of Hearing Loss
   1. Treatment of hearing loss in children
   2. Medical and surgical treatment of hearing loss, except in otosclerosis
   3. Fenestration

V. Remedies
   1. Speech reeducation
   2. Lip reading
   3. Hearing aids
   4. Rehabilitation and reemployment
   5. Social, psychologic and educational aspects
   6. The audiology center

B. *Practical Courses and Demonstrations*

I. Tone and Speech Audiometry—Basic and Special Methods

II. Selection of Hearing Aids

III. Fenestration: Training on Specimens (This course will be arranged if the amount of interest warrants it. Reservations can be made for only a few participants. A special fee will be charged to cover the costs of specimens, instruments, etc.)

The number of teachers required will naturally depend on the number of physicians enrolling for the courses; hence the following list should not be regarded as final:

Bijtel, Amsterdam
Bordley, Baltimore
Carhart, Chicago
Cawthorne, London
Dishoeck, Amsterdam
Ewing, Manchester
Frenckner, Stockholm
Furrer, Zurich
Glorig, Washington
Groen, Utrecht
Hardy, Baltimore
Holmgren, G., Stockholm
Holmgren, L., Stockholm
Huizinga, Groningen
Knapp, Boston
Larsen, Odense
Lempert,* New York
Littler, London
Lund, Copenhagen
Lüscher, Basel
de Marè, Karlstad
Peyser, Stockholm
Rueds, Zurich
Shambaugh, Chicago
Siirala, Åbo
Simson-Hall, Edinburgh
Sourdille, Strasbourg
Stevens, Harvard
Tato, Buenos Aires
Tegnér, Stockholm
Vetter, St. Gallen
Zwislocki, Basel

*According to information just received, Dr. Lempert will bring with him as teachers Dr. Meltzer, Dr. Weaver and Dr. Dorothy Wolff.

The committee in charge of the course are Gunnar Holmgren, Torsten Skoog, Paul Frenckner, Lennart Holmgren and C. A. Tegnér, secretary.

Applications for enrolment, together with the deposit, should be forwarded to the secretary (33, Birger Jarlsgatan, Stockholm C, Sweden; telegrams, CATEGNER, Stockholm), who will keep the applicants informed about the progress of the arrangements. Further information of general interest will be published in each number of *Acta Oto-Laryngologica*. Applicants who wish the committee to reserve hotel accommodations for them should state the price range and type of room, whether it should be single or double and with or without bath, the date of arrival and the probable date of departure.

Abstracts from Current Literature

Ear

SPECIAL TECHNIQUES FOR THE DIAGNOSIS AND TREATMENT OF PSYCHOGENIC DEAFNESS. W. G. HARDY, Ann. Otol. Rhin. & Laryng. **57**:65 (March) 1948.

The incidence of psychogenic deafness is not known. Army and Navy reports suggest an incidence of from 10 to 20 per cent among personnel on active duty who were hospitalized for treatment of severe disability in hearing. It is difficult to evaluate the influence of military environment and even more difficult to correlate military and civilian situations. In many cases in military centers the etiologic picture has shown no direct relation to military environment, and the problems involved were such as might be encountered in daily living. Accurate diagnosis is the first concern.

Assuming that psychogenic deafness is a psychosomatic disorder, the clinician must be able to differentiate psychologic accompaniments of organic deafness from psychogenic involvement, which operates causally in such a way that some maladjustment of the personality is referred to the ear itself. Contrary to some commonly held beliefs, failure to evoke the emotional conflict, or failure to demonstrate improved hearing in a single session with intravenous injection of a barbiturate, is by no means uncommon and does not necessarily confirm organic deafness. No single factor in the clinical findings is clearly indicative of a psychogenic involvement in a case of hearing disability. Psychogenic deafness may range, in causal terms, from malingering (as a symptom of behavior maladjustment) to hysteria (in which the conversion symptom is an involuntary expression of emotional conflict at the subconscious level). The classic hysterical deafness seems to occur relatively infrequently. The psychogenic overlay on organic disease is by all means the commonest type of psychogenic hearing involvement.

The diagnosis is the responsibility of the otologic clinician, for his special knowledge of the pathology and physiology of the hearing mechanism must be brought to bear on a wide variety of clinical data. He is responsible for determining the consistency of test findings and the deviation from classic patterns of otic pathologic processes.

Routine audiometry, both pure tone and speech, are done and repeated to check variations. The Stenger and the Lombard tests are done and also the Doerfler-Stewart test, which is possibly more specific in isolating the malingerer and the psychogenic patient. Although the latter test can only suggest a possible ratio between psychogenic and organic involvements, it is reasonably specific in determining the presence of voluntary or involuntary repression of the hearing function. These routine and special tests show evidence of dissimilar conditions but do not clear up the question of the ratio between psychogenic and organic dysfunction. Hypnosis is suggested for this, and once the patient is sufficiently relaxed the response to both pure tones and speech is checked. Once it has been determined that the hearing threshold is better than routine tests showed, the clinician has evidence, even if not quite exact, of the relation between psychogenic and organic dysfunction. It is safe to assume that the thresholds so obtained indicate the maximum of organic impairment.

When there is a psychogenic overlay on organic disease, there is no harm in employing posthypnotic suggestion of improved hearing. It is wise, too, before the patient is awakened, to suggest amnesia for the hypnotic experience. The key to

therapy, once the proportion of organic involvement is known, is the ability of the patient to gain insight into the relation between the causal factors and the symptoms. In many circumstances this phase of the treatment can often be more readily accomplished by the otologist than by the psychiatrist.

Twelve patients, 2 of them controls, are presented to illustrate the use of the technics described. MILLER, Philadelphia.

THE USE OF ROENTGEN RAYS IN SMALL DOSAGES FOR THE RELIEF OF PAIN IN INFLAMMATIONS OF THE EXTERNAL EAR. J. W. MCLAURIN, Laryngoscope **57**:317 (April) 1948.

McLaurin has had excellent results with the use of roentgen radiation for alleviation of pain in cases of inflammation of the external ear. He uses small doses and emphasizes that relief from pain and not cure of the condition is to be expected. With one treatment there was usually enough relief from pain to allow local treatment to be instituted.

In some cases of otitis media also this therapy seemed to be of benefit.

HITSCHLER, Philadelphia.

FOREIGN BODY IN THE EUSTACHIAN TUBE: REPORT OF A CASE. ROBERT C. MCNAUGHT, Laryngoscope **58**:67 (Jan.) 1948.

A piece of slag entered a patient's ear and perforated the ear drum. Washing at the time probably forced the foreign body to its resting place, the external end of the eustachian tube. Otorrhea followed. The metallic piece was discovered by roentgenogram. It was removed by shrinking the eustachian tube and applying pressure to the pharyngeal orifice, by means of a catheter, blowing the object out into the external auditory canal.

A bibliography and review of the few cases reported on of foreign body in the eustachian tube is included. HITSCHLER, Philadelphia.

A CASE OF OTOSCLEROSIS. GUNNAR HOLMGREN, Pract. oto-rhino-laryng. **10**:230, 1948.

The author discusses a case of otosclerosis in which the fenestration operation has been performed. Despite the fact that the fenestra ovalis of the patient remained open for nine years, there was no improvement in hearing acuity. Holmgren states that the failure of the patient to improve was due to closure of the round window. PERSKY, Philadelphia.

HYDROTYMPANUM IN THE DIFFERENT CLINICAL ASPECTS OF THE TUBO-TYMPANICAL CATARRH. H. A. E. VAN DISHOECK, Acta Oto-laryng. **36**:429 (Dec.) 1948.

The author states that in tubotympanal catarrh fluid in the middle ear is caused by the combined effect of negative pressure and inflammation. However, even the maximal negative effect of —60 cm. of water alone cannot produce a transudate. Only very high negative pressure, such as occur in aerotitis, can cause this. On the other hand, chronic tympanic catarrh without negative pressure, as in the case of an open tube or perforated ear drum, produces either no or very little fluid.

In acute cases the fluid is formed by active secretion. In these cases the negative pressure, if present, acts to promote secretion; yet it is diminished by the increasing accumulation of fluid and the swelling of the membranes. In the healing

phase of acute catarrh the fluid is resorbed despite the consequent increasing negative pressure; this indicates that negative pressure alone cannot produce the fluid. ROBERT LEWY, Chicago.

Pharynx

CONTRIBUTION TO THE STUDY OF LATENT TUBERCULOSIS IN THE LYMPHATIC RING OF WALDEYER. F. LANGRAF, Pract. oto-rhino-laryng. **10**:555, 1948.

At the sanatorium for children at Leysin, Langraf operated on 448 children, with a total of 689 operations. These children were presumably normal and healthy. He had a routine histologic examination made of both the tonsils and the lymphoid tissue that had been removed from these children, and he found that 13.3 per cent of the lymphoid tissue and 17.9 per cent of the tonsils showed the presence of tuberculosis. However, in spite of these findings, removal of tonsils and lymphoid tissue does not cause any danger to the child. Tuberculosis of the lymphoid tissue is frequently associated with tuberculosis of the palatine tonsils. These tonsils either may be decidedly hypertrophied or, when there is no hypertrophy, may be accompanied with other forms of tuberculosis, such as glandular or pleuropulmonary. Tuberculous involvement of Waldeyer's ring is usually of hematogenous origin. Primary tuberculosis of the pharynx is extremely rare.

PERSKY, Philadelphia.

BEHCET'S SYNDROME: REPORT OF A CASE. K. FAABORG-ANDERSEN, Acta oto-laryngologica **36**:146, 1948.

The author reports a typical case of Behcet's syndrome and discusses some of the other cases mentioned in the literature. The patient had the typical triad which, according to the author, consists of recurring aphthae of the mouth and the pharynx, recurring ulcerations of the genitals (in this instance lesions were found on the posterior portion of the scrotum) and recurring uveitis associated with hypopyon. The latter condition may in time develop into blindness.

The disease lasts for many years, and no form of therapy yet known is effective. It is noted that it occurs most frequently in areas of the mucous membrane where minor or other traumas have occurred. The disease is believed to be caused by a virus, but that this is true or that any other specific etiologic agent is the cause has not yet been established. ROBERT LEWY, Chicago.

Nose

INTRANASAL APPLICATION OF BUFFERED TRIPLE-ENNAMINE (PYRIBENZAMINE) SOLUTION IN ALLERGIC RHINITIS. J. BREM and J. ZONIS, J. Allergy **20**:70-73 (Jan.) 1949.

An initial trial of a 2 per cent tripelennamine hydrochloride solution (pyribenzamine®) in cases of chronic perennial rhinitis caused pronounced nasopharyngeal irritation, and a solution of 0.5 per cent strength was substituted. Using this 0.5 per cent solution in 81 patients with allergic rhinitis, the authors concluded that over 90 per cent of the group obtained satisfactory results. No serious local or general effects were noted. Topical therapy was found to be effective when antihistaminic drugs orally administered could not be tolerated because of drowsiness and other distressing symptoms which they produced. An additional advantage in the intranasal use of this medication was the absence of rebound phenomena. Local effects, however, are temporary, and the usual allergic diagnostic and therapeutic measures must be pursued. FRIEDBERG, Chicago.

Miscellaneous

RESPIRATORY AIR FLOW PATTERNS IN CHILDREN. ROBERT KAYE, JAMES L. WHITTENBERGER and LESLIE SILVERMAN, Am. J. Dis. Child. **77**:625 (May) 1949.

In the Harvard School of Public Health, and in various children's hospitals, the authors sought to determine the patterns of air flow with an instrument called the pneumotachograph, which is illustrated in the original text. They state that a beginning has been made by this means on a study of respiratory diseases in children in spite of the limitations of fright, etc., on the part of small children, especially those under 4 years. However, some measurements were satisfactorily taken of "minute volume." A clearcut alteration in the general pattern of respiratory flow was seen in children with asthma, severe bronchiectasis, kyphoscoliosis, and respiratory disease associated with fibrosis of the pancreas. The abnormal pattern was like that in adults with silicosis, emphysema, asthma and extensive fibrosis due to healing in pulmonary tuberculosis. In 6 patients with paralysis of the intercostal muscles after poliomyelitis there was a decided alteration in the pneumotachogram. The paper is illustrated with 11 figures.

VOORHEES, New York.

STREPTOMYCIN THERAPY IN TUBERCULOUS MENINGITIS. ABRAHAM LEVINSON, Am. J. Dis. Child. **77**:709 (June) 1949.

Levinson reports a series of 19 cases from the Children's Division of Cook County Hospital and the Hektoen Institute for Medical Research. The patients ranged in age from 6 months to 13 years; 10 were boys and 9 were girls; 15 were Negro, 3 were Mexican and 1 was white of unstated nationality. Streptomycin was given intramuscularly every three hours for ninety days. At the time of reporting, 8 of the patients were still alive (1 for fifteen months, 1 for thirteen months, 1 for twelve months, 1 for eleven and a half months, 1 for eight months, 1 for five months and 2 for three months after the start of therapy). Levinson states that he found intrathecal administration of streptomycin unnecessary in treatment of tuberculous meningitis.

Encephalitic symptoms were frequent and sometimes overwhelmed the meningitic symptoms. Deafness was ascribed to the meningitis in 1 patient; in another, streptomycin may have injured the hearing. Occasionally the erythrocyte count was increased to 6,000,000 or over, and relative eosinophilia was noted, usually in the third month. The cerebrospinal fluid was xanthochromic in 5 children; in all patients the cell count and the protein level rose at the beginning of treatment and then fell; glucose values rose, fell and then gradually became normal. The streptomycin level of the blood three hours after administration varied from 0 to 160 units; the spinal fluid concentration varied from 0 to 320 units.

Levinson concludes that the word "cure" is not properly used in connection with streptomycin treatment of tuberculous meningitis, since relapses may occur at any time and may end fatally; however, in view of the almost 100 per cent mortality associated with previous methods of treatment, "all patients with tuberculous meningitis should be treated with streptomycin."

In the discussion of this paper, W. W. Briant Jr. (Pittsburgh) cites the fact that in a recently reported series of cases of miliary tuberculosis the mortality rate for children was 80 per cent; the 20 per cent salvage was credited to streptomycin. Failure of streptomycin to cure tuberculous meningitis may be due to the inability

of the drug to control miliary tuberculosis. Rapidity of control at the original site of the hematogenous infection has much to do with the outcome. Improvement in a special area (the meninges) means little if the "feeder area" is controlled poorly, or not at all. It appears that the incidence of neurologic complications is considerably greater with intrathecal administration than with intramuscular administration, without a corresponding increase in survival rate to justify intrathecal administration. Use of 10 to 20 mg. of streptomycin per kilogram of body weight may be adequate or it may not, but it is questionable whether the dose per kilogram per twenty-four hours is as important as the duration of treatment in estimating the toxicity of streptomycin. In the absence of complications, the dose should be large enough to accomplish the desired result.

G. J. Levy (Memphis) cites a recent statement of Corper and Cohn (*J. A. M. A.* **137**:357 [May 22] 1948) that streptomycin cannot be considered to cure human tuberculosis, since it does not destroy virulent tubercle bacilli or completely retard their development in vivo. In experimental tuberculosis, Corper and Cohn noted that streptomycin did retard the disease but could not prevent a lethal outcome. Levy agrees with Levinson regarding the great value of streptomycin in the treatment of tuberculous meningitis and the desirability of its administration in every case. "Before the advent of streptomycin," he states, "there was universal agreement that tuberculous meningitis carried 100 per cent mortality."

VOORHEES, New York.

CLINICAL AND EXPERIMENTAL STUDIES WITH ORTHOXINE IN THE TREATMENT OF BRONCHIAL ASTHMA. J. J. CURRY, J. E. FUCHS and S. E. LEARD, J. Allergy **20**:104 (March) 1949.

This study was undertaken to test the action of certain recently synthesized sympathomimetic drugs which were thought to possess relatively little pressor action or stimulating effects on the central nervous system. Of the group, beta (ortho-methoxyphenyl) isopropylmethylamine hydrochloride (orthoxine®) appeared to be effective in relieving asthma.

Clinical and experimental studies of orthoxine® and ephedrine sulfate were made in two groups of patients with bronchial asthma. It was shown that orthoxine® in doses of about 200 mg. was comparable to ephedrine sulfate in doses of 30 mg. in protecting the asthmatic subject against the reduction in vital capacity and asthma-like attacks due to histamine or methacholine chloride administered parenterally.

Clinical studies with the two drugs indicated that in the doses used they were equally effective in relieving mild cases of asthma. However, side reactions were much less frequent after the use of orthoxine,® and there was little or no pressor or stimulating effect on the central nervous system. It was concluded that orthoxine® has a definite place in the management of bronchial asthma, because it is an effective bronchodilator that may be given orally and is followed by relatively few undesirable side reactions.

FRIEDBERG, Chicago.

PRACTICAL TECHNIQUES IN SPEECH TRAINING FOR CLEFT PALATE CASES. CHARLOTTE G. WELLS, J. Speech & Hearing Disorders **13**:71 (March) 1948.

Sequence in training for velar-pharyngeal port closure, to be modified in each case, is recommended for consideration. The area and parts involved should be identified in each case. The person with cleft palate must be aware that he is

attempting to gain control of the velar-pharyngeal area. The steps in the general procedure for developing nasopharyngeal port control are tongue exercises, voluntary movement of the palate, with mirror observation, stimulation of the palate by air pressure and directional breathing exercises. Games, such as blowing at ping-pong balls and at toys made of matchbox tops, are useful. Rubber tubing is used to carry sound from the nostril to the ear. No work in cases of cleft palate can be considered successful until it carries over into sequential speech. No person with cleft palate can be really helped until personal, physical, educational and social problems have been met. PALMER, Wichita, Kan.

PERSONALITY CHARACTERISTICS AND EMOTIONAL PROBLEMS IN STUTTERERS UNDER THE AGE OF FIVE. PHILIP J. GLASNER, J. Speech & Hearing Disorders **14**:135 (June) 1949.

Seventy stutterers under 5 years, studied relative to their emotional and social maladjustments, were divided into three main types: a healthy, aggressive type of child, with emotional pampering and criticism from a relative suddenly coming into the family; an ungainly, awkward type, with brilliant parents and a whole constellation of emotional disturbances because of hostility from one parent or the other, and a dependent, confused and fearful type. The author states that children of this type have a long background of overprotection and pampering and overanxious, excessively perfectionistic parents. PALMER, Wichita, Kan.

NEURO-ALLERGY IN CHILDHOOD. T. WOOD CLARKE, New York State J. Med. **48**: 393 (Feb. 15) 1948.

Pediatricians are stressing the importance of allergy more and more, but they are not yet fully conversant with its implications in the meninges and the brain. Three characteristic results of an allergic reaction are smooth muscle spasm, edema of the brain coverings and increased glandular secretion. An increase in the spinal fluid or giant urticaria of the brain or its coverings may cause symptoms resembling those of brain tumor. Headache, vomiting, dizziness, failing vision, paresis, convulsions and psychotic changes in the personality are notable symptoms of increased intracranial pressure. Sick headaches may be due to cerebral allergy. Conditions of the optic and auditory nerves, particularly the so-called Ménière's syndrome, may be due to neural allergy in children and adults. Peripheral neuritis due to serum sickness may come on twelve to twenty-four hours after injections of tetanus antitoxin, involving the fifth and sixth cervical nerves and causing paralysis lasting from three months to a year; it is sometimes permanent. Prompt recognition and treatment with epinephrine and the intravenous injection of hypertonic solutions of sucrose or dextrose are said to reduce the edema and so relieve the pressure on the nerves.

Two hundred cases of infantile convulsions were studied by J. Thompson in London; he believed they were due to "anaphylaxis," stating that continuing the offending diet might lead to epilepsy in later life. Diet affects neural sensitivity in some persons. Cow's milk is prominently mentioned in this respect. The asthmatic child often shows nervous excitability but improves on a change of diet from one too rich in offending proteins.

Clarke says that no allergist suggests that all cases of headache, maladjustment, etc., are due to allergy, but that enough of them ought to make one consider an allergic condition as a cause. VOORHEES, New York.

SURVEY OF RAGWEED POLLINATION IN THE NEW YORK METROPOLITAN DISTRICT IN 1947. EUGENE H. WALZER, BERNARD B. SIEGEL, ROBERT A. CHAIT and MATTHEW WALZER, New York State J. Med. **48**:2019 (Sept. 15) 1948.

The data for this study were obtained through the division of allergy, Jewish Memorial Hospital, in Brooklyn, and the report was made to the New York Allergy Society, May 6, 1948. The first extensive survey was undertaken in 1946, in collaboration with the Pollen Committee of the American Academy of Allergy. Annual surveys are planned for the coming years. Within a radius of 50 miles (30.5 kilometers) samples of air containing pollen were taken and were found to be comparatively low for 1947, as compared with 1946. Seasonal totals for New Brunswick and Teaneck were higher than the average for New York City! But at Verona, Garden City, White Plains, Fire Island and Maplewood the totals were less than the average for New York city. At this time, the value of the efforts to exterminate weeds (ragweed) cannot be fully estimated, chiefly because of the relatively light pollution of the air. The technic used was recommended by the American Academy of Allergy. Slides were thinly coated with petrolatum U.S.P., exposed to the air for twenty-four hours, then stained with Calberla's solution and the pollen grains counted under low power magnification (cover slip, 4.84 sq. cm.). Approximately 1,000 slides were counted by two of the authors, who checked each other's observations. VOORHEES, New York.

PASSAGE OF A FOREIGN BODY (FOXTAIL) THROUGH THE BRONCHUS, LUNG, PLEURA AND THORACIC WALL. M. M. SEHRING and E. B. SHAW, Pediatrics **1**:210 (Feb.) 1948.

A 4 year old boy was first seen with fever and abdominal pain of twenty-four hours' duration and was observed to have signs of pneumonia of the lower lobe of the right lung. Four days previously he had swallowed some grass. At first the mother stated that this provoked no coughing, but later she insisted that it did produce a paroxysmal cough immediately.

The child responded to early sulfathiazole therapy; but during the continuation of this treatment his temperature rose to 41.3 C. (106.3 F.), at which time he was admitted to the hospital and given penicillin therapy. On his entry to the hospital, the roentgenogram showed signs only of pneumonia of the lower lobe of the right lung without evidences of atelectasis or of pleural fluid. The temperature promptly dropped to and remained normal, but ten days after he first ingested the grass a fluctuant swelling appeared in the right midaxillary line in the sixth intercostal space. Aspiration of the swelling removed 3 cc. of pus and a foxtail which was 5 cm. in length. Thereafter, all signs of inflammation rapidly disappeared, and the chest, judging from the results of physical examination and its appearance on roentgenograms, remained entirely normal.

The case is reported because of its unusual nature. A similar case was reported by Seydell in 1937, who found 8 similar cases reported from 1879 to 1936. No further cases have been discovered in the literature. At no time were evidences indicative of the necessity for bronchoscopy, and, because of the property of self propulsion of the foreign body, its removal could have been accomplished only when it appeared in the subcutaneous abscess.

FROM THE AUTHORS' SUMMARY [AM. J. DIS. CHILD.].

PRESENT METHOD OF MENINGITIS TREATMENT IN CHILDHOOD. G. FANCONI, Schweiz. med. Wchnschr. **78**:121 (Feb. 14) 1948.

The improvement in the prognosis of meningitis during the last nine years is remarkable. The use of the sulfonamide drugs in meningococcic meningitis has given satisfactory results. Large doses of the sulfonamides in influenzal meningitis have been equally satisfactory, and the cure of the previously fatal cases of pneumococcic meningitis with the use of penicillin by either injection or the intrathecal route constitutes a third success. The use of streptomycin in tuberculous meningitis, while not always successful, does in many instances postpone a fatal issue for many months. In meningococcic meningitis Fanconi uses a sulfonamide drug, 0.3 to 0.2 Gm. per kilogram of body weight six to eight times a day, until the patient is free from fever. After three days half this dose is given by mouth or intramuscularly. He advises administration of large quantities of fluid by mouth, subcutaneously or with the drip method by vein. In the septic forms he uses large doses of the sulfonamide drugs and penicillin intravenously and intramuscularly, and in some cases intrathecally. Influenzal meningitis is nearly always fatal. However, since the massive doses of sulfonamide drugs have been used, Fanconi reports, aside from those who came to the hospital in a moribund condition, 16 of 17 patients recovered, though 3 had residual defects. As already indicated, he uses large doses of sulfonamide drugs, large quantities of fluids and intravenous infusion of normal saline solution or dextrose solution and streptomycin in the most resistant cases. Fanconi is of the opinion that penicillin treatment is not of noticeable value in influenzal meningitis. In pneumococcic meningitis, especially in infants, Fanconi stresses the importance of penicillin used intramuscularly or intrathecally and of the administration of sulfonamide drugs, with a large fluid intake, blood transfusion and fluids intravenously. In his opinion it is important to use penicillin intrathecally in pneumococcic meningitis. In this disease, as well as in influenzal meningitis, treatment should be continued in some instances for weeks or months, as relapses may occur again endangering the life of the patient. He calls attention to the fact that the intrathecal use of penicillin may cause meningeal reactions such as muscle spasms or hemorrhages in the brain or subdural spaces. He states that not more than 10,000 units of penicillin well diluted should be used in intrathecal injections. He again calls attention to the fact that streptomycin is powerful, though not always successful, in the treatment of tuberculous meningitis. He is of the view that if the disease could be diagnosed early more could be accomplished.

ABT and ABT, Chicago [AM. J. DIS. CHILD.].

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

William A. Smiley, M.D., President

Lawrence J. Lawson, M.D., Secretary

Monthly Meeting, March 7, 1949

Electroencephalography in Diagnosis of Hearing Loss in the Very Young Child. Dr. Richard E. Marcus, Dr. E. L. Gibbs and Dr. F. A. Gibbs (by invitation).

Hearing in the young child may become seriously impaired or lost completely as the result of numerous local or systemic diseases and certain, poorly understood, hereditary or intrauterine influences. Because of the tremendous social handicap imposed on the child with defective hearing, an early, definitive diagnosis is imperative, so that specific therapy and proper educational technics may be employed.

This study is concerned with the nonspecific arousal response noted on the electroencephalographic tracing when a loud sound is used as a stimulus while the child or infant is asleep. If the sound is perceived, a definite change in the brain wave pattern becomes evident. Stimulation of olfactory and taste sensation was used as a control in 19 patients whose ages ranged from 17 months to 9 years. Thirteen of these patients had no previous demonstrable hearing and 6 had reduced hearing but defective speech. Four of the 13 children considered to have no hearing showed an electroencephalographic arousal response to loud sounds. An incidental, but potentially important, finding was that of intracranial damage, as shown on the electroencephalographic tracing. The finding of intracranial damage may indicate that some of these children do not have true "peripheral" hearing loss but may actually have one or more types of auditory aphasia. Further studies for calibration of the sound stimulus and correlation of intracranial damage with hearing loss are contemplated.

DISCUSSION

Dr. Thomas C. Galloway: Were any of these children tested with conditioned reflexes? This method, developed by Dr. C. A. Aldrich, seems of value with infants. A bell is rung, and then the child is gently pricked with a pin. After a few such trials the child with hearing cries as soon as the noise is made.

Dr. Sherman Shapiro: Might not the depth of slumber and the sedation used influence the degree to which the patients responded?

Dr. R. D. Russell: I have been under the impression that substances like acetone and ammonia produced fifth nerve responses. I should like to know how Dr. Marcus separates that from olfactory response.

Dr. Richard E. Marcus: In reply to Dr. Galloway, we did not do such testing. The children were so profoundly deafened that any conditioning one would attempt would have to be prolonged—using pins or other methods which parents would object to; whereas with the method we used there is no difficulty.

The child may put up a fight while the electrodes are being placed, but soon falls into a nap. It is an innocuous procedure. I do not know what more we could learn from the conditioning technic with the profoundly deafened child, except that it might be worth while for comparison.

In reply to Dr. Shapiro, the relation of response to the depth of slumber and sedation has bothered us. Drs. Gibbs have done about 20,000 electroencephalographic tracings, in a fair percentage of which the children have been under barbiturate-induced sleep. They believe that the effect of barbiturates can be evaluated separately, unless sedation has been carried to the point of anesthesia, in which instance the arousal response to sound cannot be obtained. The depth of sleep, however, is important, and we try to keep the children at the level of light sleep. The depth of sleep can be judged from the type of electroencephalographic tracing. Some of the children fall into deep sleep, from which they can be aroused only with the greatest difficulty; it is sometimes necessary to shake them to get them awake. When this occurred, the absence of the arousal response to sound was discounted; another record was accomplished under more favorable circumstances, or else the child was not used for the purposes of this study.

Dr. Russell asked about the use of acetone. Acetone may stimulate sensory systems other than that of olfaction alone, but it does not concern us directly since we were looking for a method of stimulation other than auditory. If the child responded to olfactory or tactile stimulation and not to auditory stimulation, we concluded that he could not hear.

Evaluation of Irradiation of Pharyngeal and Nasopharyngeal Lymphoid Tissue. DR. FRANCIS L. LEDERER.

Considering all available evidence, irradiation of the nasopharynx is without danger. It should, however, be used with restraint because of the incomplete knowledge of radiobiology and the empiricism of the present dosage.

It is not a substitute for surgical procedure, and its use should be confined to cases of conduction deafness associated with the presence of lymphoid hyperplasia in and about the orifices of the eustachian tube. Transitory hearing loss, allergic manifestations and infection in the nose, sinuses or nasopharynx must be recognized and treated accordingly. The use of irradiation in irreversible hearing deficits is to be deplored.

The importance of the past history with relation to previous irradiation is stressed because of the possibility that a repeated series of treatments may produce untoward tissue changes; the danger to the technician himself in the use of this therapy is also stressed.

Histologically, all stages of degeneration in the lymphoid tissue may be demonstrated after irradiation. Thereafter, varying degrees of reparative change follow, from granulation tissue to localized areas of ossification, in occasional cases. No further metaplastic change has been noted, nor has there been any alteration of the surface epithelium.

DISCUSSION

DR. JOHN R. LINDSAY: This subject came up for discussion at the 1949 annual session of the American Medical Association. In my discussion of Dr. Farrior's paper, I pointed out that in one's experience in examining the eustachian tube histologically, lymphoid tissue in the eustachian tube proper, in anything more than microscopic nodules, is rare. On the other hand, the tissue in Rosen-

muller's fossa extends up along the tube to a degree greater than is usually realized, 1 cm. or more beyond the level of the torus. One should not expect an effect from beta rays for, I am quite sure, they would not reach that distance. On the other hand, lymphoid tissue extending into the mouth of the tube does not occur beyond 1 or 2 mm. I was interested to hear Dr. Guild confirm this observation.

If we physicians are trying to get at the pathologic process in the eustachian tube beyond the very mouth of the tube, we are probably dealing with something other than hyperplastic lymphoid tissue. The thesis of the essayists at the annual session of the American Medical Association was to recommend roentgenographic therapy in such cases. If one uses roentgenographic therapy for such pathologic conditions in the tube, one is not treating hyperplastic lymphoid tissue but probably the result of former inflammatory reactions.

Did Dr. Lederer wish to imply that the existence of cartilage and bone in the tonsil demonstrated was the result of irradiation? Cartilage and bone have long been known to occur relatively frequently in tonsils, and there would be no reason to attribute their presence in this case to the radiation.

Dr. G. H. Mundt: No one is competent to say that he does or does not get satisfactory results. I should differ with Dr. Lederer on one point: if a radium applicator is to be used, I think the otolaryngologist should apply it.

Some of us have used radium as honestly as we know how. We have made repeated audiograms on children and watched them carefully, with special attention to the pharynx. Long before radium was advocated I frequently removed large masses of lymphoid tissue from the nasopharynx in children who had had adequate tonsil and adenoid operations. In some, the improvement in hearing and general condition was most satisfactory. My experience with roentgen rays, in the treatment of tonsils and adenoids has not been satisfactory.

Dr. Lederer spoke of inadequate operations. The faucial tonsils can be taken out satisfactorily, but no one can be certain how much recurrence there will be following surgical removal of the lingual and pharyngeal tonsils—and, by the way, the word adenectomy should be eliminated. Some children receive pronounced benefit and some obtain little if any improvement, just as with any method of treatment.

Dr. Thomas C. Galloway: In our office my associates and I have used radium often in hearing impairment of patients referred from a school survey. We came to its use reluctantly, but the results have been about 80 per cent favorable. There were some side effects that were especially good, including help for allergy, as Crowe has pointed out.

The favorable record of roentgenographic therapy in malignant conditions is impressive. There are a number of explanations of its action, and doubt exists whether it acts on the protoplasm, the nucleus or the blood supply. In infection, there is doubt about what irradiation actually does. It probably acts to destroy infiltrating white cells and free enzymes, and I am sure it is of considerable value. I have reported 100 cases of infection, carefully studied, in which irradiation was used with much benefit.

I agree with Dr. Mundt that radium can be a carefully applied modality, and in the hands of the trained laryngologist it is possible to apply it accurately; if properly applied it does not disseminate infection. I think that it cannot be dismissed lightly or condemned.

Dr. Francis L. Lederer: I had always assumed that cartilage and bone could be found in any tonsillar material that was sectioned, their presence not being specifically identified with changes as a result of irradiation. In this particular

tonsil, radon was actually placed in the tonsil; whether that had any relation to the presence of bone and cartilage in the tonsil, I do not know. Dr. Tamari did not identify cartilage and bone as a normal observation in tonsils, and he believed it to be the result of irradiation per se. I believe that cartilage and bone is a normal finding in the irradiated structure of the tonsil. The technic should be adequate for depth. This was a quotation from Dr. Wachowski, and otherwise I am in agreement with what Dr. Lindsay has said. I had hoped that the audience would help me with my indecision, and I thought I would hear some testimonials on the therapy. I have no more proof than Dr. Schulz and Dr. Robbins have, and I am not willing to say that this is a dangerous treatment in proper hands. However, I contend that otorhinologists have no business using radium unless they are qualified in its use, and I think that the average man does not know enough about it. Some men make no differentiation of radium as an element and radon. I have heard about wonderful results publicized by others, but I cannot boast about my own.

Blood Pressure Changes in Fenestration Surgery. Dr. M. J. Tamari and Dr. M. H. Cutler.

Blood pressure changes were studied in 68 patients with a diagnosis of otosclerosis who had been selected for fenestration surgery. There were no cardiovascular diseases in the group, and emotional types of patients were not included. As a result of this study, we concluded that depression in blood pressure does occur after traumatic irritation of the labyrinth. Blood pressure checks on persons with complete loss of labyrinthine function did not reveal any changes after caloric or rotatory stimulation. Lowering of blood pressure occurs in characteristic types which depend on the preoperative blood pressure level. The depression is most manifest one hour after the operation has been completed and generally persists for from one to three days, independent of other labyrinthine reflexes and reactions.

DISCUSSION

Dr. Sherman Shapiro: This excellent work confirms what has been well known experimentally, and I think it deserves more than passing interest. I should suggest, for the sake of completeness, that a series be carried out with a group of patients undergoing plastic surgery with local anesthesia to serve as controls to show the difference of procedure; the same type of medication might be of value.

Dr. Alfred Lewy: Is there any comparable list of other cases to serve as controls in which surgical operation was performed with local anesthesia? Is this more or less a shock phenomenon?

Dr. Max H. Cutler: Someone asked how much influence the preoperative medication had. The medication was started eight hours before operation and was rather light. Pentobarbital sodium and morphine were used, and we felt that while it did tend to lower the blood pressure it was not sufficient to cause this particular change.

We have performed endaural radical mastoid operations with the patient under local anesthesia, and in no instance has there been a strikingly low blood pressure. However, in 1 case, during operation, the anesthetist called attention to the sudden drop in blood pressure. The following day that patient had definite nystagmus and symptoms of labyrinthitis, entirely due to the operation. We hope to continue the study.

William A. Smiley, M.D., *President*
Lawrence J. Lawson, M.D., *Secretary*
Regular Monthly Meeting, April 4, 1949

Development of the Otic Capsule in the Region of Surgical Fenestration.*

Barry J. Anson, Ph.D.

Continuing the study of capsular development, we are now able to account for the chief steps in ossification of that portion of the otic capsule which is the site of surgical fenestration.

The observations are based on the 30 series of sections selected from more than 300 in the otologic collections at the University of Wisconsin and Northwestern University Medical School. From the 30 series, 8 were chosen for description and discussion of ossification.

OBSERVATIONS

In the fetus of 126 mm. (16 weeks) the otic capsule is still almost completely cartilaginous. However, at the level of surgical fenestration, the first evidence of ossification appears in the region external to the ampulla of the lateral semicircular canal.

By way of prediction, it may be said that development of the capsule in the region of the lateral canal, where the surgical fenestra is made, is different from that of the remainder of the capsule with respect to two features: (1) On this portion of the capsule the head of the growing incus exerts pressure, and (2) here the expanding epitympanic recess and antrum produce an early erosion and thinning of the capsular wall, with subsequent rebuilding.

In the 150 mm. (18 weeks) fetus, periosteal bone is present in the cochlear portion of the capsule, appearing in its apical and basal areas. In the greater part of the canalicular portion of the capsule, the cartilage remains unaltered. However, on the tympanic wall of the lateral semicircular canal, osteogenesis is, on the contrary, precocious. In the process of ossification of the tympanic wall of the lateral semicircular canal, toward the ampulla of the semicircular duct, the capsule shows internal, intermediate and external layers. The region that was originally occupied by cartilage is largely excavated and filled with fetal marrow, except for a few islands of the original cartilage which, as part of a middle layer, are being converted into intrachondrial bone.

At this point it should be explained that the otic capsule is composed of three layers of bone; these are, from the outside inward, the periosteal; the endochondral, with contained intrachondrial remnants, and the endosteal. The inner layer is actually periosteal bone, although the term endosteal is commonly employed.

The outer layer is formed from the cambium layer of the periosteum around the cartilaginous capsule. It is like the periosteal layer of a typical long bone. The inner layer is always thin and fairly uniform in structure. It is derived from the endosteum, or inner periosteum, and constitutes the immediate wall of the labyrinthine spaces. The middle layer is the most complex in structure; it is

* This study was carried out in collaboration with Dr. Theodore H. Bast, Department of Anatomy, University of Wisconsin.

A full-length illustrated paper (of which the present text is an abstract) will be submitted to an otologic journal for publication.

composed of islands of altered cartilage (intrachondrial bone), which become surrounded by endochondral bone. As a result of the spread of endochondral bone, the primitive marrow spaces are virtually obliterated.

The tympanic wall of the lateral semicircular canal is the site of ossification center 3. The first center appears in the 126 mm. (16 weeks) fetus, the last, or fourteenth, in the 183 mm. (21 weeks) fetus. The maximum size of cochlea, canals and of surrounding capsule is thereupon attained.

Even in a fetus of 193 mm. (22 weeks), the external layer of the lateral canalicular wall has begun to undergo erosion, despite the fact that it has been so recently formed. Moreover, part of the subjacent middle (endosteal) layer is being removed.

At 210 mm. (23 weeks) endosteal bone has formed a complete shell for the labyrinthic spaces (cochlea, vestibule and semicircular canals); cartilage persists, as a large mass, only in intercanalicular territory, lateral and superior to the subarcuate fossa. Since no epiphysial areas are present (as secondary centers of ossification), no further growth of the capsule will take place. This is equivalent to saying that the capsule has attained substantially adult dimensions and that enlargement can hereafter be only peripheral, through the thickening of the periosteal layer. The latter stratum is, at this stage, in a state of active increment in the cochlear portion of the capsule, but in the canalicular portion it is limited to the antral area. Between the inner and outer layers, the middle zone is one of quiescence; here the intrachondrial bone is well formed. Since the endochondral bone is just beginning to appear, the middle layer is still loosely textured. Looseness persists until a period just prior to birth when, owing to rapid deposition of endochondral (replacement) bone, the capsule will become solid, or "petrous." At this stage in the tympanic wall of the lateral canal, erosion has resulted in the removal of all of the perichondrial bone, some of the endochrondral bone and the embedded intrachondrial bone.

In the capsule of the 270 mm. (30 weeks) fetus, in the cochlear region, periosteal bone has developed rapidly, to add extracapsular bulk to the fetal temporal bone. Increment deepens the internal acoustic meatus and adds length to the cochlear portion of the capsule. The endosteal layer is thin and remains so throughout life. Replacement bone (i. e., of the middle layer) is experiencing a surge of development in the canalicular portion of the otic capsule. The "spicules" of this middle layer are a combination of the two types of osseous tissue, namely, the replacement bone and the altered osseochondral islands, of antecedent formation. Comparable activity is postponed in the cochlear portion until the fetus has reached the stage of term or during late prenatal life. This is tantamount to saying that during approximately a ten week period, terminating at birth, the production of endochondral bone progresses rapidly in the territory of the semicircular canals and vestibule, then to be shifted to the cochlea. In the canalicular wall there is evidenced a filling in of the middle layer in its deeper portion, with concurrent and continued erosion on the superficial aspect. The outer layer is now entirely wanting.

In a specimen of term fetus, in the cochlear portion of the capsule, the inner layer is fused with the middle stratum; the latter is now composed chiefly of endochondral bone, cartilage islands being inconspicuous. The structure of the capsule immediately around the vestibule is similar to that of the cochlea. However, the intercanalicular area is still nonpetrous, being made of endochondral bone, with relatively sparse remnants of cartilage islands. In the region of surgical fenestration, the middle layer is undergoing condensation, but owing to erosion the entire wall is thin.

In a 6 month infant, in the cochlear region, on the cranial (medial) aspect of the temporal bone, marrow spaces are abundant in the periosteal layer. However, in the deeper stratum of the same layer, the periosteal shell of the cochlea (that is, the original capsule) remains petrous in character. Comparably, in the canalicular region, periosteal bone has been removed in favor of antral bone, which presses into the intercanalicular tissue. On the tympanic surface of the lateral semicircular canal, the thin inner layer is still distinguishable from the middle layer. The middle layer is very erose and irregular. On this tympanic surface new bone is being deposited; seemingly, it is periosteal in nature but is of secondary formation.

In a 19 year adult, with the exception of the tympanic wall of the lateral semicircular canal, the histologic structure of the bone is (allowing for individual variation) essentially similar to that of the fetus at term. The bone of the lateral canalicular wall is now quite compact. Intrachondrial bone is represented by a few scattered islands. The tympanic margin of the wall is smooth. New bone has been deposited to one-half the total thickness. Consequently, the thickness of the wall in this region merely equals that of the 150 mm. fetus.

COMMENT AND CONCLUSIONS

The otic capsule differs from all other skeletal elements of the human body with respect to function, speed of development, schema of ossification and histologic fabric.

Functionally, the otic capsule serves as an osseous box to house protectively the organs of hearing and equilibrium.

The development of the capsule is dramatically rapid; it attains adult dimensions when the fetus has reached the middle of intrauterine existence, whereas a typical long bone continues growth through a period of twenty or more years.

Constitutionally, the otic capsule is unique. It is made up of numerous (totally, 14) ossification centers. Bone spreads almost concurrently from the core of each of the 14 originally independent centers not, as in a typical long bone, from a point of initiation in the middle of a diaphysis (shaft) toward terminal epiphyses (at the extremities). Fusion of centers is peripheral. There exist between the expanding centers no zones of secondary or "epiphysial" growth, such as regularly occur in a long bone. Fusion, with complete obliteration of fusion lines between contiguous centers, early converts the otic capsule into an osseous unit, a single bone; in this respect it differs from the cranium, whose constituent elements (parietal, frontal, etc.) remain at least partially separable along their boundaries of sutural contiguity.

In relation to histologic fabric, the otic capsule is exceptional. Within its substance a considerable fraction of its primordial cartilage is retained throughout life, in the form of chondral tissue embedded in endochondral bone. Its middle layer, once formed, persists; it is never remade into haversian tissue. Its outer layer, thickened to outbulk the contained capsule, subsequently becomes pneumatized through invasion of petrous bone by mucous membrane.

The outer, periosteal, layer of the cochlear portion of the capsule is complete before the twenty-eighth week (246 mm.). In the canalicular part of the capsule it appears later (21 weeks, 183 mm.); it spreads beyond the limits of the original capsule, toward the mastoid region; it continues to expand, as an extracapsular addition, until the time of puberty. The inner, or endosteal, stratum is the simplest; it forms a mere shell for the labyrinthic canals, cochlea and vestibule; the layer is complete at midterm (approximately 183 mm. stage) and thereafter undergoes

no considerable increase in dimensions. The middle (intrachondrial and endochondral tissues combined) is the most complex in structure. In both cochlear and canalicular divisions of the otic capsule, intrachondrial bone appears in the fetus of 18 weeks (147 mm.); these cartilage islands attain maximum distribution within approximately six weeks after their first appearance (that is, at 23 weeks, or in the 210 mm. fetus). Around the cochlea they retain primordial form for almost a month (up to the stage of 27½ weeks, or 246 mm.). Thereupon, slow formation of endochondral bone is initiated and the cartilage islands become covered by a thin layer of the endochondral bone—without other alteration of the "spicules" on which deposition of the replacement bone takes place. At term, the process is suddenly accelerated, continuing through the first few years of infancy with such alacrity that the cochlear region comes rapidly to resemble that of an adult bone. In the canalicular region there is no such initial lag in development. From the first, the deposition of endochondral bone around the canals is rapid up to the stage of 7 or 8 months (30 weeks, or 270 mm.), at which stage adult condition is attained. Activity begins in the region of the superior semicircular canal; the areas of the posterior and lateral canals follow.

The intercanalicular part of the capsule, composed chiefly of endochondral bone, never becomes fully converted into bone of petrous nature. It retains marrow spaces, which may persist in the adult ear or be replaced—beginning in the twenty-eighth week (240 mm. stage)—by pneumatic spaces which spread from the tympanic antrum. Except for the intercanalicular portion of the capsule, pneumatization is restricted to bone of periosteal nature, which was added peripherally to the primordial osseous capsule. In other words, except in the region named, the process of pneumatization leaves the capsule in substantially the form in which it existed in the fetus at midterm.

In the surgically important region of the otic capsule, namely, the tympanic wall of the lateral semicircular canal, the process of ossification is, in the early stages, the duplicate of that which operates to form any typical portion of the capsule: The three layers of bone are produced, to replace the primordial cartilage; together they undergo no expansion after once having fused along their contiguous surfaces. However, at approximately the stage of midterm, the process of ossification in the region of surgical fenestration begins to follow a special course. The capsule here consists of the three regular layers in the fetus of 18 weeks (150 mm.). At 22 weeks (193 mm.), the recently formed periosteal bone is being removed. So rapid is this process that in a 30 week (270 mm.) fetus, not only has the outer layer been resorbed, but varying amounts of the middle layer are removed, with some of the constituent cartilage islands. Following this phase of resorption, new bone is added onto the tympanic surface to restore a smooth contour. As a consequence, the mucoperiosteal membrane, instead of the usual periosteal layer, covers this secondary replacement bone.

DISCUSSION

DR. GEORGE SHAMBAUGH JR.: Dr. Anson has continued his original and monumental studies of the development of the labyrinthine capsule with this lucid, detailed and beautifully complete account of the horizontal semicircular canal. The clinical application of such fundamental research is not always evident, but in the case of the labyrinthine capsule there are important clinical implications.

As Dr. Anson has so clearly shown, the middle or endochondral layer of the labyrinthine capsule is unique in the rapidity of its formation from fetal cartilage during the fifth month of fetal life, reaching adult dimensions long before birth

and retaining its histologic form without further alteration throughout the life of the person. It is this inert quality that helps to make possible the creation of a permanently open fenestra in a high percentage of operations. In a recent study of 300 consecutive cases of fenestration operation, in which the patient was followed for more than two years after operation, loss of the hearing improvement, presumably due to osseous closure, occurred in less than 1 per cent. The surgical technic that has yielded this result includes the following features:

1. Endochondralization of the fenestra (the wide exposure of the relatively inert endochondral layer by removal of the more active periosteal layer far beyond the margins of the completed fenestra, which is left on top of a dome-shaped elevation). The endochondral bone is easily recognized through the operating microscope by its yellowish color and the coarsely granular appearance imparted by the islands of ossified cartilage. The endochondral bone has been likened to pineapple ice cream, as compared to the smoother appearance and white color of the periosteal layer, resembling vanilla ice cream.

2. The microimmaculate removal of every particle of bone dust, with the help of continuous irrigation and the operating microscope.

3. Endosteum-hemming of the fenestra (the careful preservation of the intact endosteal membrane up to the sharp knife edge of the fenestra, so that it will unite with the periosteum of the skin flap). Thus, there is left no naked, raw, unprotected bone edge, from which osteogenesis will occur. The use of a knife to outline a bony lid, as recently described by Lempert, may have the disadvantage of stripping the endosteum and leaving an edge of unprotected bone.

4. The fenestra to be epidermis-periosteum gloved (as thin a skin flap as possible should hug the bony margins of the fenestra as tightly as possible). The endochondralization technic, leaving the fenestra on top of a dome, ensures this glovelike fit.

5. Burnishing of the bone around the fenestra with a smooth, hard gold burr (to help to inhibit osteogenesis, possibly by sealing the openings of the bone capillaries).

6. The prevention of fibrosis by a painstaking aseptic technic combined with perfect hemostasis. This step is important, for fibrosis in the perilymph space is frequently followed by osseous invasion of the fibrous tissue.

I should like to inquire about one point not mentioned by Dr. Anson. Of the 2,100 consecutive fenestration operations performed at Northwestern University Medical School, an otosclerotic focus involving the horizontal semicircular canal was observed at operation in 62, an incidence of about 3 per cent. The otosclerotic focus in the endochondral layer is easily recognized under the operating microscope by its whiter, softer, more vascular appearance, as contrasted with the hard, yellowish endochondral bone with its islands of ossified cartilage. In some cases the otosclerotic area in the semicircular canal is continuous with the area around the anterior end of the oval window; but in others, the focus in the bony horizontal semicircular canal is discrete, with no connection with the focus at the site of predilection. I should also like to ask Dr. Anson if he or Dr. Bast has encountered any areas of unossified cartilage in the semicircular canal region that might act as a nucleus for the development of such otosclerotic changes.

Book Reviews

Recent Advances in Oto-Laryngology. Second edition. By R. Scott Stevenson, M.D., Ch.B., F.R.C.S.; Surgeon, Metropolitan Ear, Nose and Throat Hospital; President, Section of Otology, Royal Society of Medicine. Price, $6. Pp. 395, with 114 illustrations. The Blakiston Company, 1012 Walnut St., Philadelphia 5, 1949.

Stevenson's book is an excellent review of the recent advances in otolaryngology. It is concise, well written and unbiased. The writer appears to be well aware of the good work being done here and abroad. The chapter on audiology is exceptionally well done and includes information that every otologist should have.

Lempert and Shambaugh are given commendable mention for their fenestration work, and Proetz and Hilding share the spotlight for their important work on the physiology of the nose.

The antibiotics, including the sulfonamides, penicillin and streptomycin are handled succinctly and in proper proportion to the specialty as a whole. In general, this volume, although not a textbook, presents in a scientific, conservative manner the latest and most authentic papers in otolaryngology.

Atlas der Augenkrankheiten. Fifth edition. By Rudolf Thiel, M.D. Price, $18. Pp. 225, with 634 figures, mostly in color, and an index. Grune & Stratton, Inc., 381 4th Ave., New York 16, 1948.

This book is the fifth edition of a standard German atlas of diseases of the eye, which was first published in 1936. It was not intended to be a complete textbook on ophthalmology but to serve as a reference book for the general physician, who may be called on to treat diseases of the eye. The fact that five editions have been published indicates that it is popular among German-speaking physicians.

The colored photographs and drawings are up to the usual prewar German standard. The subject material is organized in atlas form, beginning with the lids, lacrimal apparatus and orbit and proceeding to the eye from the outside inward, in logical sequence. Grouped together in the discussion of each disease are the pertinent anatomic and physiologic points and short, etiologic, diagnostic and therapeutic suggestions. Wherever applicable, photographs are included of microscopic sections of the involved tissue.

The beauty of the illustrations is enough to recommend this book, even to those who have little or no knowledge of the German language. For the specialist in ophthalmology, the book will be a worth while addition to his library, chiefly because of the illustrations. For those physicians whose practice includes some medical ophthalmology, it will be a valuable and quick reference for diagnosis and a review of salient anatomic and physiologic considerations.

Fundamentals of Otolaryngology: A Textbook of Ear, Nose and Throat Diseases. By Lawrence R. Boies, M.D., Clinical Professor of Otolaryngology; Director of Division of Otolaryngology, University of Minnesota Medical School, and associates. Price, $6.50. Pp. 443, with 184 figures. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1949.

This book is dedicated to Dr. H. P. Mosher, of Boston, who has so often and for so long set the pace for teaching subjects on the ear, nose and throat. The names of his associates—Connor, Hilding, Hilger, Hochfilzer, Holmberg, Phelps,

Priest and Tangen—are mentioned on the title page. Each of them has taken a special chapter for his province and has told the beginner nearly all he needs to know.

Some items suffer from being too sketchy. For example, furunculosis of the external auditory canal is allotted less than one page, and the treatment for it less than ten lines. Since this is a condition commonly seen by the general practitioner and is sometimes as important to the patient as is an impending mastoiditis (for which it may be mistaken), it is insufficiently discussed. Moreover, few such conditions are cleared by "incision under gas." In fact, incision may "spread" the infection unless followed up with careful antisepsis. Furunculosis of the vestibulum nasi is also an important condition, since it can become fatal by extension through the veins to the brain. One fails to find this condition mentioned in the index or text.

In general, however, the book is well planned. A chapter on applied anatomy and physiology of the ear, nose and throat heads each section, respectively, and lays the groundwork for what follows. The book is satisfactory as an aid to clinical instruction of medical students.

Pollen Slide Studies. By Grafton Tyler Brown, M.D. With a foreword by Wallace M. Yater, M.Sc. (Med.), M.D. Price, $6. Pp. 122, with 98 illustrations. Charles C Thomas, 301-327 E. Lawrence Ave., Springfield, Ill., 1949.

Pollen in plants is analogous to spermatozoa in man. Pollen may be useful in propagating plants, but it is a pest to some animals, especially man. The weather has much to do with scattering pollen, especially wind and rain, but pollen is not destroyed by frost, as many think. In fact, some experiments were made in freezing and thawing pollens and they showed that the pollen did not lose potency through freezing. This book contains 182 drawings of pollen grains and fungus spores, with 34 photomicrographs. It is a manual, a textbook, a reference work and an atlas combined. The blurb states that anyone who can read and knows how to use a microscope can "count" pollen and prepare extracts for testing by following the information given here. "Allergy as a specific field of medicine has finally come into its own." The public uses the term, however, indiscriminately, but perhaps not harmfully. Ear, nose and throat specialists are always being told about "my sinus trouble," and in many instances there is no sinusitis, per se, but a stuffiness of the nose and general malaise because of allergy. Psychosomatic medicine has repeatedly declared that much of so-called allergy is not due to pollens at all but to a reaction of the sympathetic nervous system to environment, particularly of persons to whom jealousy, hatred and other ruling passions play such a large part in creating unhappiness. Pollen slide studies, of course, have nothing to do with that phase of respiratory illness. This book contains much of value for those interested in "pollenosis."

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26, Mich.
Place: San Francisco. Time: June 28-30.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 8-13, 1950.

American Broncho-Esophagological Association

President: Dr. Leroy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-26, 1950.

American Laryngological Association

President: Dr. Ralph A. Fenton, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Hotel Mark Hopkins, San Francisco. Time: May 23-24, 1950.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-27, 1950.

SECTIONS:

Eastern.—Chairman: Dr. Robert L. Moorhead, 125 Remsen St., Brooklyn, N. Y.
Southern.—Chairman: Dr. William D. Stimson, 899 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. J. Marion Sutherland, 3001 W. Grand Blvd., Detroit.
Western.—Chairman: Dr. J. B. Naftzger, 6777 Hollywood Blvd., Hollywood, Calif.

American Otological Society

President: Dr. Philip E. Meltzer, 20 Charlesgate, W., Boston.
Secretary: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 3, N Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 21-22, 1950.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Samuel F. Kelley, 47 E. 61st St., New York 21.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. George E. Shambaugh Jr., 55 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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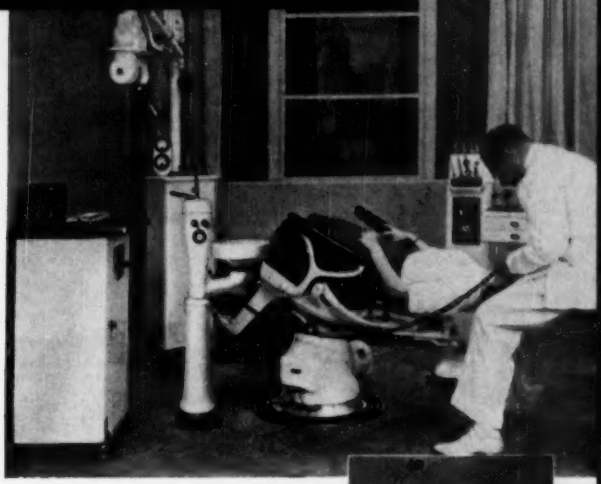

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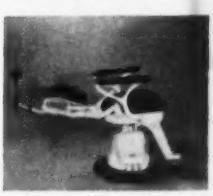

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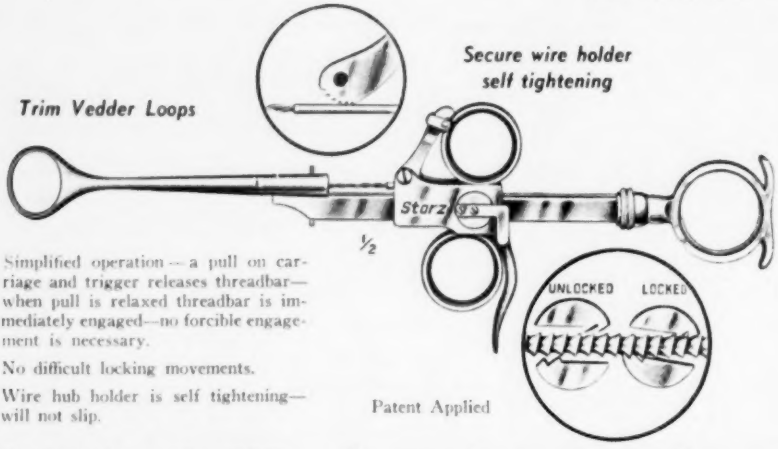

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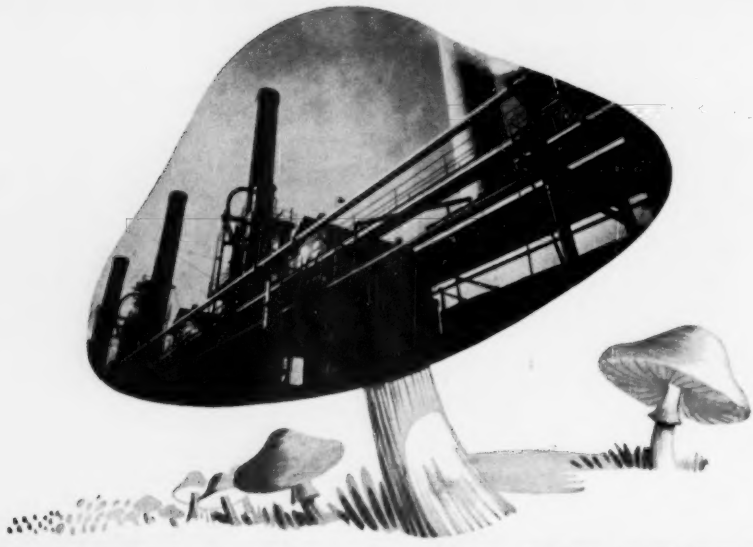

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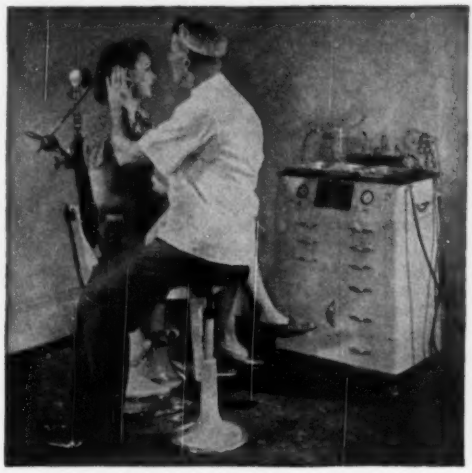

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